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MDCCCXCVII.

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1863. Stark, J., M.D., Bridge of Allen, Stirlingshire, N.B.
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1863. Rowe, T. Smith, M.D., 1, Union Crescent, Margate.
1881. Russell, E. G., M.B., B.Sc., Surg. Lieut.-Col., I.M.S. Bengal Army.
1859. Sanderson, J. Burdon, M.D., F.R.S., 64, Banbury Road, Oxford.
1884. Simpson, W. J., M.D., Bengal Club, Calcutta.
1854. Spinks, C. N., 3, Palmyra Square, Warrington.
1889. Street, A. F., M.D., St. Mildred's Road, Westgate-on-Sea, Kent.
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1894. Tebb, W. Scott, M.D., Boscombe, Bournemouth.
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1853. Valerj, —, M.D., Rome.
1855. Virchow, Rudolf, Professor, 10, Schellings Strasse, Berlin.
1856. Von Iffland, —, Beaufort, New Quebec.
1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.

TRANSACTIONS

OF

The Epidemiological Society of London.

INAUGURAL ADDRESS OF SESSION 1896–97.

On the Endemic Prevalence of Infective Diseases in the Tropics.

By Professor J. LANE NOTTER, M.A., M.D., President.

(*Read: November 20th*, 1896.)

Gentlemen,—Permit me to thank you most sincerely for allowing me the privilege of presiding over your deliberations. It is indeed a high honour to be elected President of the Epidemiological Society, on whose roll are the names of able and distinguished men whose labours have indeed added much to our knowledge of disease. I cannot hope that it was any personal claim that induced the members of this Society to select me for the position I occupy this evening, but rather from a desire to recognise that branch of the profession to which I have the honour to belong—whose service under varied conditions of climate and races brings them ever in contact with diseases which afford opportunities for epidemiological study. It is with some diffidence that I have accepted the position, as I am fully aware of the responsibilities attached to the presidential chair; and not the least has been my difficulty in selecting a subject for my address to-night, which I could hope to present in a manner at all worthy of the occasion. I trust, therefore, the subject I have chosen will not be thought to be foreign to the aim of the Society: it is one of great interest to the military surgeon, as well as to those of our profession whose lot is cast to live and combat with disease in tropical and sub-tropical climates.

It is a well-recognised fact that certain diseases prevail in an endemic form within a measured range of distribution over the earth's surface, and from these endemic centres epidemics spread, either in isolated groups, or devastating campaigns which have claimed its numberless victims.

For example: cholera, until the early part of the present century was endemic, not only in India, but limited to a few districts in that country, and these districts still remain the endemic home of the disease. Plague is endemic in Mesopotamia and Egypt, and has been so for ages—certainly for two centuries, if not more, before the Christian era. Yellow fever is limited to well-defined geographical limits: the shores of the Gulf of Mexico, including the West Indies and part of the Atlantic coast of the United States. On the other hand certain diseases, such as enteric fever, etc., know no geographical limits, and the general distribution area of these diseases shows that mere questions of country or climate have little or no influence on their occurrence.

Let us now consider the question more in detail, and in the first place with special reference to cholera.

What are the causes which excite or determine an endemic home for this disease; or can we trace any special characteristics which produce in its endemic area a favourable environment for its growth and multiplication?

Although accurate accounts of the disease only go back to 1817, a study of the history of cholera shows that it has with each successive pandemic wave obtained a wider geographical distribution, and that its area of occasional prevalence covers the greater part of the inhabited globe. Throughout this history there runs the important assumption that the disease did not arise *de novo* at each and every place where it has shown itself, but rather that it is indigenous at certain points on the earth's surface from which it tends to extend to other parts.

Further, there can be now no reasonable doubt that the home or place of origin of cholera is to be sought for in India; and that, thanks to the labours of Bryden, Macpherson, Macnamara, Bellew and Hirsch, with many others, in regard to the proper habitat of cholera in India, our information points to Lower Bengal as the true headquarters of the disease, with possibly Bombay and Madras as also endemic foci. Further, the history of the disease shows that its occurrence beyond these limits has always been associated with a conveyance or introduction of the specific cholera poison, and moreover, that the diffusion of the disease in those places is not so extensive, or in proportion to the importation of the poison; or, in the words of Hergt, the rise of a cholera epidemic at any one place implies, besides importation of the contagium, certain local conditions. These local conditions may be either general sanitary defects, peculiarities of climate, or of soil. It is

mainly to these two latter features that I wish to direct your attention this evening. On these grounds, therefore, it is from its own particular home or endemic region that we must largely seek evidence in regard to any connection between the origin and diffusion of this disease and soil.

At the outset, a remarkable fact has been noted, namely, that cholera has always attained its widest diffusion and its greatest intensity in those localities distinguished by a certain physical soil character, namely, permeability to water and air, and on those kinds of rock which have a large capacity for retaining the moisture which has fallen upon them. As far back as 1832, it was noted that places with a rocky bottom enjoyed a relative if not absolute immunity from the disease—be it in India, in Russia, Canada, or Nova Scotia. This dependence of the diffusion and origin of cholera upon geological and physical characters of soil was first clearly pointed out by Bombée, who indicated that on its appearance in Europe, cholera spread with the greatest rapidity over those tracts of country with an alluvial or tertiary bottom: and that, on the other hand, it made very slow progress over soils belonging to the older formations, especially the primary rocks. In 1854 this view was upheld by the behaviour of an epidemic in the Pyrenees: where all the localities with a granite bottom were spared, but that immunity ceased whenever a layer of alluvium or débris, however thin, overlaid the rock. Similarly, Brittany and Poitou, which rest on the primary rocks, the department of the Loire and the Auvergne, which are on secondary formations, have been exempt, while the population on the tertiary and the alluvial soil of the Gironde and Rhône were decimated. The writings of McClelland, and Lewis, and Cunningham reveal that similar observations are available from India, especially that lateritious soil of that country upon which severe epidemics of cholera have raged, is highly porous. On the other hand, cholera may occur on a rocky bottom, wherever the latter is disintegrated by the weather, or broken up or covered by detritus, and so made liable to become sodden, like the permeable rocks of a newer formation.

Drasche and others have quoted the occurrence of epidemic diffusions of cholera in Malta as instances of the disease spreading upon a rocky and hard-bottomed soil; but the limestone rocks of Malta are in the main of a very soft and porous kind of stone, which sucks up moisture like a sponge: likewise, the physical properties of the alluvial soil of Bengal and other parts of India have been clearly

demonstrated to be of a highly porous nature, and in a remarkable degree retentive of moisture. In considering, then, the incidence of cholera upon any particular soil, it is not the geological character of the soil itself, but the saturation dependent thereon, in which the true explanation of the phenomena is largely to be sought; but even this does not cover the whole case, for it again is governed by soil heat, rainfall, the subsoil water level, the soil and general climatic influences, to say nothing of the nature and quantity of organic matter in the soil; each and all of these contingent factors need to be separately examined in regard to cholera prevalence. There appears to be no doubt that a relatively high temperature, both of air and soil, materially favours the production of cholera; in the case of air, this is not shown so much by the prevalence of the disease at times of maximum temperature, as in the absolute abatement or extinction of an epidemic with a fall in atmospheric temperature.

On the other hand, from facts which have been slowly and laboriously collected concerning the soil temperature in various parts of India, where cholera is more or less endemic, we find that a high temperature of the soil corresponds differently, yet more closely, with the course of cholera prevalence than does that of air. The credit of the earliest and most systematic attention to this point belongs to Lewis and Cunningham who, in 1876, made a most valuable series of observations on soil temperature in Calcutta. They found that at a depth of 6 ft. from the surface a soil heat of between 78° and 79° Fahr. corresponds with the maximum prevalence of cholera, and that the soil temperature at Calcutta is higher than that of the atmosphere during the cold season. Further and later observations show that any marked excess or falling short of that temperature, for any length of time, appears inimical to manifestations of the disease, while a continuance of that heat for a few days seems equally potent in initiating an outbreak.

So much for soil heat; but as concerns soil moisture as affected by rainfall, we find that in Calcutta, as well as in all that part of Bengal which belongs to the so-called true endemic area of India, the maximum of cholera sickness falls in the dry season (October to May), and more particularly in that portion of the dry season which is at the same time the season of greatest heat (March to May). The chain of circumstances in Bombay is very similar: there also we see an abatement of the disease occurring at the

setting in of the rain, and an increase of the epidemic during the dry season (December to June), more particularly in the very hottest part of that season of the year (April, May and June). In Madras one of the two maximum periods begins in January (end of rainy season) and lasts till March; the other develops when a certain quantity of moisture falls in July, and lasts till the setting in of the heavy rains in October. In the non-endemic area in India we find some points of resemblance, and some of contrast, to those offered by the endemic area. The whole history of cholera, both in India and elsewhere, abounds with or suggests inconsistencies in its behaviour with reference to rainfall and moisture. They will appear less glaring and unintelligible if we bear in mind that rainfall, as a causal element, is purely indirect through the soil. While a certain degree of soil moisture appears to be an essential condition for the occurrence of cholera as an epidemic, so absolute dryness, or complete saturation of the ground, seems to hinder the development or bring an epidemic to an end. Soil moisture is not a question of so much or so little rainfall, but also of the physical character and saturation point of soil, to say nothing of the state and level of the subsoil water. It is obvious that when the subsoil water level stands at a low level, copious rains will produce an effect altogether different from that of moderate rains, when the subterranean water level is high, and *vice versa*: and that these results again will be still further varied according to whether the soil is a porous one (sand) or a moderately porous one (loam or marl). Chiefly owing to the writings of Von Pettenkofer, much atttention has been given to this question of subsoil water level and its bearings on cholera diffusion, particularly in India, where Lewis and Cunningham made systematic observations for eight years in and around Calcutta. Their researches show that there the average water level for the entire year is 12.4 ft., and that the average annual fluctuation in level is 6.4 ft. The averages show that the water is nearest the surface in September, and therefore falls steadily until it reaches a maximum depression in May, from which it again rises rapidly. There is a considerable amount of coincidence apparent between the course of the phenomena of water level depression and cholera prevalence: so much so that the period of maximum prevalence coincides with the main part of the period of maximum depression of the water level, and one of the months of minimum prevalence with the month of minimum depression. When, however, the data are more closely

examined, the coincidence is found to be a general one only, and numerous divergencies between the courses of the two phenomena present themselves. On the whole, Lewis and Cunningham found that though the prevalence of cholera in Calcutta is associated with a low level of the soil water, the data clearly indicate that the absolute water level in itself is of no importance. The subsoil water level must be looked at in the light of an index to the changes in soil humidity, from mere moisture to actual saturation of the overlying soil strata, as well as influencing the question of soil ventilation.

In regard to this latter factor, although our methods of estimating it are imperfect, and with reference to non-epidemic localities our data are meagre, still, in the true endemic area, as Lewis and Cunningham indicated, degrees of soil ventilation seem to bear a direct relationship to cholera prevalence; and, moreover, offer a clue to the connection of this disease with soil.

We have so far discussed certain broad physical facts existing in, or belonging to, soil during cholera prevalence, with a view to discover, if possible, what are the soil states or attributes favourable or essential to outbreaks of that disease. The evidence offered has been drawn partly from the endemic home of cholera in Lower Bengal and other parts of India, and partly from other countries in which it has occasionally prevailed as an epidemic. If we ignore the latter as being too scanty and unreliable evidence, the only soil states which appear to bear any degree of constancy to cholera incidence are: (1) porosity and permeability; (2) an average soil heat of 80° Fahr. at 4 ft.: a low level of subsoil water. This clears the ground somewhat, but it remains still to inquire what evidence is offered by the soil itself as to the existence in it of what all unite in considering does exist—the specific organic cause of cholera. Elaborate microscopic examinations of soil from various parts where the disease exists, both endemically and epidemically, have been made, but mainly with negative results.

Though the comma bacilli have been rarely found in soils, yet many observations have been made regarding their behaviour both in water and soil. All experimental facts seem to indicate that the so-called choleraic commas are extremely feeble in the struggle for existence when gaining access to ordinary soil, and tend rapidly to die out. This question, however, is not quite so simple as this would seem; and is further complicated by the fact that the more

one studies this subject the more it appears that in all probability there is a plurality of species of comma-shaped bacilli, and that these do not behave uniformly in either water, soil or any other media. We may not be dealing with distinct species, but with races or modifications due to change of environment. As Dr. Adami puts it, there must be some little latitude in our conception of species among the bacteria; we must be prepared to discover considerable variations in the properties of any one species. By a due appreciation of this dictum it is probable that many experimental inconsistencies may be explained. The existence of more than one comma in different cholera dejecta is probably dependent upon the varying conditions of the individual patient's intestinal heat—analogous to the variation in symptoms and mortality of cases, as well as to variations in value of certain remedies in the disease.

It does not infrequently occur that, during an epidemic, cases present themselves which, both in respect to symptoms and mortality, must be looked upon as choleraic, but in which the intestinal contents are devoid of cultivable commas, or indeed of commas at all. Such may be merely cases in which the commas have failed to find intestinal conditions in which they could breed true: just as outside the body the same commas can, under the influence of artificial external conditions, assume important and more or less persistent morphological and physiological properties.

These considerations open up the whole controversy as to whether cholera commas in their life-history undergo change of form, or whether they can assume a resting or spore stage, in which, though overlooked and regarded as absent, their germs are all the while merely waiting suitable external conditions to renew their well-known shape and effects. The more the life-history of this comma bacillus is studied, the clearer it becomes that this microbe is not one of the ordinary schizophyta; or if it be, then it is merely an evolutional phase of some higher organism, or, what is more likely, a true parasite existing in man and animals, and needing another host, possibly the soil itself, wherein to complete its life-history. Experiments made with various samples of soil, including ordinary alluvial sand from the Punjab, alluvial marl from Lower Bengal, the laterite clay from the North-West Provinces, the black soil from the Deccan, detritus from the Maltese limestone, and garden loam from England, all show that cholera commas do rapidly and completely die out in soils when either very dry or absolutely saturated with water; or when exposed

in soils, however dry or moist, to a temperature below 60° Fahr. or above 85° Fahr., or when mixed in soil with putrefactive matter, or with fæcal matter.

This incapacity on their part for continued life in such circumstances is due to want of oxygen, excessive cold or heat, and to the pressure of fungi and saprophytic forms of life hostile to them. On the other hand, in moist states of soil short of saturation, and in media offering conditions short of those just enumerated, mere inability to discover commas after a lapse of time as commas, is no proof of their destruction and disappearance, as they are capable of undergoing morphological changes, and assuming a resting or spore stage in which their duration of life is indefinite, and from which, on being transferred to more congenial surroundings, *e.g.*, the alimentary canal of man and animals, they can assume active properties and powers.

A consideration of these facts justify the conclusion that, provided a soil be porous or permeable to air, free from putrefaction and free from any excess or marked deficiency of water, commas can live and multiply in it for an indefinite time. The chemical or geological features of soil are immaterial to the life of comma bacilli, while the impurities apparently most favourable to their life-history in soil seem to be those derived from vegetable feeders. In the endemic area the soil in the main is not the essential site of the processes and changes resulting in the production of the organism which in man produces cholera. The soil concerned in these changes is that layer lying above the water level, or first impermeable stratum in a locality, and once seeded with a specific organism, the development in the soil, or diffusion from the soil, of the disease depends on certain conditions of that layer. These conditions are: permeability to air-moisture not exceeding 60 per cent. nor less than 20 per cent., a mean annual temperature of 72° Fahr., the limits at 3 ft. being 50° and 85° Fahr., a moderate amount of contained organic matter, an absence of decomposing or putrefactive processes, and an absence of human or animal excreta in any excess. Any locality presenting these conditions throughout the year may be said to be capable of affording an endemic habitat for the cholera organism. Lower Bengal, as typified by Calcutta, fulfils these conditions. Assuming that prevalence in that area is a fair test of production, we ought to find, if these conclusions are correct, that the amount of specific material developed increases with the mass of generating stratum: this is exactly what we do find, as the maximum and

minimum of cholera prevalence in Calcutta coincides with the maximum and minimum lowering of the water level; or in other words with the maximum and minimum of non-water-logged soil. If we go a step farther, and equally assume prevalence in the same area to be an index of diffusion, then there are two main channels by which a material developed or harboured in the soil may reach human beings in any locality: these are the water and the air occupying the soil interspaces. The phenomena of seasonal fluctuation in prevalence fail either to explain or support the idea of the water being the channel of diffusion; were it so, the maximum prevalence ought to occur at that period when the meteorological conditions are calculated to facilitate the entrance into the drinking water of materials derived from the bodies of those suffering from the disease. June, July, August, and September are the periods when most material is washed into the tanks and drinking-water supplies, yet these are the months of minimum prevalence. So, too, in May, the rainfall is heavier than April, yet instead of an increased there is a decreased prevalence; while in November more cases of the disease occur than in October, though the latter month presents greater chances of inwash of material by rain into the tanks and wells than the former.

If, however, we regard the air as the channel of diffusion by which the cholera poison passes from the soil to the subjects of the disease, we find that the facts are different. Exactly in accordance with this hypothesis, maximum soil ventilation occurs during March and April coincidently with the maximum of prevalence; and the minimum of soil ventilation occurs during the rainy season, which is the period of minimum prevalence. Strong as is the evidence in favour of the diffusion by soil air emanations and dust, yet it does not cover all cases, as shown by the lessened incidence of the disease on all places provided with a good water supply. Calcutta is a case in point: the disease has certainly lessened there, but has not disappeared. The existence of a pure water supply has reduced the number of cases, by reducing the facilities for the propagation of the disease, by virtue of a wholesome drinking water being substituted for one polluted by the recent dejecta of the cholera-stricken. This experience, without appreciably weakening the soil theory of the disease, merely strengthens the belief that possibly both channels of diffusion are at work, and that too great reliance must not be put on either the one or the other.

These remarks apply altogether to the endemic home of cholera. How do these conclusions affect the rôle played by the soil in the non-endemic districts? All evidence points to the fact that the nearer the soil of any district, especially in India, approaches in character and conditions to that constituting the lower part of the Gangetic plain, the greater is the likelihood that cholera will be found to prevail there. This is peculiarly well illustrated in the tendency of the disease to prevail on all alluvial soils, especially near rivers, such as in the valleys of the Brahmaputra, the Nerbudda, Tapti, the Indus and Euphrates. It has been shown that the disease tends to be deferred in its seasonal manifestations, the drier the soil and climate become; so that in the Punjab, North-West Provinces, and upper parts of India, the maximum prevalence of the disease occurs just at those periods when the soil conditions most closely approach those in the endemic area of Lower Bengal when in its driest state, viz., during and towards the end of the rains. Whether the disease is not after all really endemic in several parts of India which at present are not so considered, is not very clear. It is, however, certain that personal intercourse between infected and non-infected places, while undoubtedly accounting for a very large number of cases, is insufficient to account or explain for some of the remainder, which in the main can only be explained by regarding the disease as truly endemic in hitherto unsuspected spots, and only requiring the establishment of certain indicated conditions in their superficial soil layers to determine the production of cholera: the chief of these being a drying zone of soil, already containing the specific material causative of the disease.

To summarize what has been said—

The relation of soil to cholera is peculiar, inasmuch that while its specific germ is unable to long survive in ordinary states of soil, still it can retain its vitality, increase in growth, and, too, assume a spore or resting stage on or near the surface of mud, or on banks of rivers or ponds, and in soil rich in vegetable *débris*, more or less free from putrefactive products, and in a constantly moist state short of saturation. In many of its soil needs, the cholera bacilli betray a close affinity to yellow fever; with, however, the distinction that the latter has a marked predilection for marine mud and soil, while the germ of cholera is essentially an inhabitant of fresh-water riparian soils.

It is probable that the abililty of the cholera bacilli to thrive under these conditions is not due to any special

qualities inherent in soil, since they can equally flourish in other media provided they offer the needed degrees of warmth, moisture, and nutritive matter. The most antagonistic conditions to cholera commas appears to be rapid desiccation. The close identification of the cholera bacilli with the soil is practically limited to its endemic areas, especially in Lower Bengal, where it almost exclusively exists in or on the soil by the margins of tanks, ponds, or rivers.

It is only too probable, that were these collections of water completely removed as sources of water supply for domestic use, the disease would become extinct so far as India was concerned.

Outside the endemic area, it is tolerably evident that cholera has much less relation with the soil states than it has within that area; in fact, so far as its occasional prevalence in Upper India is concerned, all cases which are not the result of direct importation from affected districts are attributable and explicable by the existence of infected foci, that is endemic centres, in the so-called non-endemic districts: these foci being in nearly every case the moist soil along the edges of ponds, tanks, and rivers.

The next tropical infective disease that I wish to bring under notice is yellow fever.

In the absence of any definite or accurate knowledge regarding the nature of the real cause of this disease, the chief evidence indicating or suggesting the special connection between yellow fever and place is derived from: (1) those facts concerning its peculiar geographical distribution; (2) those facts which show that it seldom leaves the coasts or banks of navigable rivers, periodically haunting the low and filthy quarters of seaports. The whole history of yellow fever goes to show that, though at one time and another its diffusion has been wide, still its native habitat is much less extensive. Hirsch, to whom we are largely indebted for a reliable and complete history of this disease in the past, says that: "If we disregard its isolated appearance at several points on the western shores of Europe, and its quite recent establishment as a prevalent form of sickness on the Atlantic and Pacific coasts of South America, there remains only three situations at which the disease bears an undoubted endemic character—the West Indies, the Mexican Gulf Coast, and part of the West Coast of Africa."

The first and most important point before us—if the disease is so limited in its endemicity—is this marked

localisation due to the geological characters of the soil of those parts? The answer is—practically none—for the disease has been met with on the chalk soil of Jamaica, Trinidad and San Domingo, not less frequently on the volcanic formations in St. Kitts, Guadeloupe, San Lucia and Granada, or on the alluvial soil of Western Africa, Mexico, Brazil and Peru, to say nothing of tertiary formations in Texas and the United States. In Europe it has occurred on the jurassic limestone of Valentia and the chalk soils of Cadiz; while in Sierra Leona and New England, the oldest formations, granite and the transition rocks, have not escaped yellow fever outbreaks.

Owing to the constant confusion existing in the minds of all early writers on this subject as to a probable identity of yellow fever with malarial disease, the data regarding the physical conditions of the soil in infected localities is largely unreliable. More recent investigations by careful observers indicate that the disease principally prevails along the banks and channels of rivers which are dry at certain periods, and in the low parts of seaports, particularly those abutting on or overhanging harbours, stagnant waters, or foul foreshores. Testimony of this kind clearly defines the existence of a porous, loose, and periodically saturated soil as a constant concomitant of yellow fever prevalence. No evidence appears to be available regarding the microscopic life present in soils in the yellow fever zone, nor are there any records as to the soil temperatures and soil water levels as bearing upon the occurrence of this peculiar disease in its endemic home. Allusion may, however, be made to the philosophical and remarkable theory regarding the peculiar origin of this disease as elaborated by Creighton from the forgotten writings of Audonard, and which has a direct bearing upon the connection between the affection and soil processes. The Audonard-Creighton theory of the evolution of yellow fever is that its advent into the world coincided with the rise of the slave trade, and that its habitat is or has been the ports of debarkation of these slaves. It is perfectly well known, and admitted both by Lind and Bancroft, that the slaves on the slave-ships did not suffer from yellow fever, although they suffered much from dysentery, typhus, and what used to be called "the horrors of the middle passage"; so that a slave-ship would arrive at her destination with absolutely no contagious fever on board, but yet saturated with the filth of her human cargo.

According to Audonard, yellow fever originated at all its

endemic centres from the filth of the slave-ships, which filth was the putrid dysenteric discharges of the sick negro; and that the fever owed its well-marked specific type to the fact that the matters which excited it could be traced to the negro body.

Regarded in this light, yellow fever is a "vicarious infection"—that is, it is a product from a race or class of people who have not themselves suffered from a like specific form of disease, though in the course of certain hardships and miseries imposed upon them, they have been breeding an infection for others, though not for themselves. The poison of yellow fever, whatever it may be, has been given to us in the dejecta of another race, which, brought in considerable quantity in bilges of ships to ports, has been there discharged into harbour mud and soil.

The scourings of these ships, fermenting and multiplying in the harbour and shore mud, has generated a specifically poisonous virus, which has been only too readily carried from harbour to harbour in the bilges of wooden ships; it has entered through their opening seams. and has risen in noxious vapours to infect their crews, and even delayed at times its destructive powers till it has crossed the ocean and mixed with, or obtained a footing in, the shore mud of a distant port. This view is strongly supported by the fact that yellow fever is most persistent at places where there has been least cleansing of harbours, beach and foreshore by the natural action of the tides, or where much stagnation of the harbour water exists, as in places like Havana, Port Royal, Bridgetown, and Port au Prince.

Though up to the present the isolation and identity of the yellow fever poison has not been made, still we may regard it as being essentially a mud or soil poison, and the mud or soil when it has accumulated for a time continues to be an endemic focus of the disease, and liable to periodic outbreaks in an epidemic form. Until we know more as to what is the nature of the yellow fever virus, we must leave it an open question as to how far the soil acts only as a nidus for it, or how far it exercises any specific influence on its growth or spread. Arguing from analogy, it is probable the soil only furnishes a suitable medium in which yellow fever poison may remain and multiply, and that of itself it is quite devoid of any special vitalizing influence upon the cause of this disease.

Powerful as these soil conditions are as factors in the spread and diffusion of disease germs in the tropics, we must not forget that infection and extension of diseases

can occur in other ways besides; a recollection of this fact is still more necessary in the case of those pathogenic bacteria which do not reach the soil to the same extent, or which, like the cholera bacilli, cannot be distributed from the soil in the most common manner—namely, in dust by currents of air—for the reason that they cannot withstand the necessary degree of drying. In the case, therefore, of the majority of the non-putrefactive, non-fermentative, and non-saprophytic parasites, their preservation in, and distribution from, the soil forms in reality a rare exception; and, further, the occurrence of any local and seasonal variation in the distribution of the infective diseases—which, according to the views of Pettenkofer, are only explicable on the assumption that the soil exerts an influence—does not by any means necessitate the idea of a constant connection between the soil and epidemics, nor does it exclude other modes of spread quite outside soil influence: such as by food, by water, or by direct contact, and which are for the most part more available than the indirect way through the soil.

From what has been said, there can be no doubt that pathogenic organisms do exist in the soil to a large extent; but owing to the fact that the law of the survival of the fittest holds good for microbes in the soil as for all organised things everywhere, a constant warfare and struggle for existence is going on between the pathogenic bacteria and the saprophytic microbes present in the soil: the result being that under certain conditions the power for harm of the greater number of pathogenic organisms in soil is much diminished. On the other hand, a certain number of pathogenic organisms have their special habitat in the soil, and, unlike the others, are capable of surviving in the soil, notwithstanding the saprophytes; and especially is this the case in tropical and subtropical countries.

THE SPREAD OF PLAGUE.

By JAMES CANTLIE, M.B.Aberd., F.R.C.S.Eng.;
Lecturer on Applied Anatomy, Charing Cross Hospital Medical School;
late of Hong-Kong.

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The history of plague has been so often written that it is unnecessary for me to even sketch its devastations and distribution in pre-Christian, early Christian, mediæval, or more recent times. My remarks will be confined to an exposition of matters appertaining to the epidemicity of the disease as it has been met with during the latter half of this century, and more particularly to a description of the information gathered during the outbreak in China in 1894-6, and of the most recent evidence of its activity in the Bombay Presidency. The reasons for selecting the Hong-Kong epidemic as the basis of my dissertation are, that plague was first studied there bacteriologically, and that many medical men, myself amongst the number, had ample opportunity of observing and recording the disease. Without entering into the discussion as to whether or not all epidemics styled "plague" in Biblical and mediæval literature were of the nature of the bubonic plague of recent times, there can be no doubt that since 1850 the name "plague" has been applied to a definite train of symptoms which are considered to constitute a specific disease. As we proceed it will be seen how during different outbreaks some one symptom attains a fatal pre-eminence, but not to the exclusion of others, which might at any time assume the death-lead and serve to baptise the disease as "bubonic," "hæmorrhagic," "typhoidal," "pneumonic," and so forth.

During the latter half of the century the names bestowed on plague have been as follows:—1. In 1856 in Tripoli, it was known as "typhus with glandular swelling." When the disease reached the island of Chios in the same year, it was called "petechial typhus analogous to that of Tripoli." 2. In Mesopotamia, where the disease was prevalent from 1856-85, and possibly up to the present day, the titles, "adynamic typhoid fever" and "intermittent fever with

glandular swellings" were usual. 3. In Persia, where plague seems to be endemic since 1868, the term "hæmorrhagic fever" is used to designate a pestilence which differs from the diseases with which the Persians are familiar. 4. During the years 1877-79 the Russian province of Astrakan was visited by a disease which was, and is, regarded as plague, but which was variously described as: (*a*) intermittent fever with buboes; (*b*) croupous pneumonia with buboes; (*c*) typhus with glandular swellings proving fatal by pneumonia; and (*d*) a peculiar form of mumps. That all the above-mentioned outbreaks were actually plague, a specific disease caused by a specific bacillus, will never be known; but the presumptive and clinical evidence is decidedly in favour of their being one and the same disease. 5. In Hong-Kong, where a moiety of languages obtain, the epidemic was known as:—(*a*) the "plague" or "the bubonic plague" by the British; (*b*) "la plaga" by the Spaniards; (*c*) "de peste bubonica" by the Portuguese; (*d*) "la peste" by the French; (*e*) "la pestilenza" by the Italians; (*f*) "die peste" by the Germans; (*g*) "wan-yik" by the Cantonese, the term signifying an epidemic disease of high mortality; (*h*) "yang-tsu-chwang" in Yunnan which, being interpreted, means bubonic disease; and (*i*) "li-tsu-cheng" in Pakhoi, where the disease has been endemic for many years. The name "black death" was never used, nor were any of the names by which it was known in Tripoli, Mesopotamia, or Persia, or Russia applied to it, although hæmorrhages, pneumonia, petechiæ, and typhoidal signs and symptoms were not wanting. Perhaps the most interesting name is that of the "rat plague," by which it is known in various parts of Asia, thereby affording additional proof, if such were needed, of the widespread infection of the rat during plague epidemics.

The name "plague," however, is not one which helps us to place the complaint in its proper scientific niche in the category of disease. The word conveys the idea of an unusual disease, attacking many persons and attended by a high mortality, but that gives it no scientific value any more than the "wan yik" of the Chinese. Were I to attempt to classify plague, I would place it amongst the specific fevers and particularise it under the name of "polyadenitis" and individualise it still further as "malignant polyadenitis." It may be objected that adenitis is not always present, that many plague-stricken patients die before the glands are even swollen, and that therefore the name is defective; but it is needless to point out that in

many diseases the most typical of symptoms are at times absent, and yet the cases can be classed with their kind. To justify the prefix "poly" to the word "adenitis" it is necessary to remember that, although one gland alone may be clinically apparent, most, if not all, the lymphatic glands are found to be enlarged at the post-mortem examinations. The most recent report in regard to this subject is interesting. "Thirty-three cases (22 per cent.) were received without a proper bubo. In most of these multiple adenitis was found, the general size of the glands being that of a lentil. In the latter stage of the disease, other buboes formed in different localities and complicated the case; 90 per cent. of the cases showed swelling of the intestinal, bronchial, and mediastinal glands in different stages."*

The term "bubonic", so frequently applied to the disease, is not so appropriate as would at first glance appear. A bubo is essentially the swelling of a gland caused by the entrance of septic materials in the tract of lymphatics over which the gland presides. This cannot be said to obtain in plague, for the so-called bubo of plague can in no sense be regarded as a sympathetic swelling, but merely the outcome of a blood poison. If it is to be classed a bubo at all, it must be distinctly understood to be an "idiopathic" bubo, a true "bubon d'emblée." One never speaks of the buboes of tertiary syphilis, or of scrofulous buboes. The terms may be clinically correct, but they are not in common use; they have, however, as much right to be called bubonic as the glandular enlargements in plague. With these considerations before us, I would venture to name the disease "Malignant Polyadenitis", in the hope that attention may be drawn to the naming of the disease by more competent students than myself.

Plague or malignant polyadenitis we may define as an acute febrile disease of an intensely fatal nature, characterised by inflammation of the lymphatic glands, marked cerebral and vascular disturbances, and by the presence of a definite bacillus by which the disease can be recognised scientifically when ordinary symptoms are in abeyance. In this definition no mention is made of hæmorrhage, pneumonia, petechiæ, or carbuncles.

At the present day plague is confined to Asia, but since 1850 it has made its appearance in Europe, Asia, and Africa. The *western* limit of plague during the latter half of the century is the Canary Islands, where it raged in 1852;

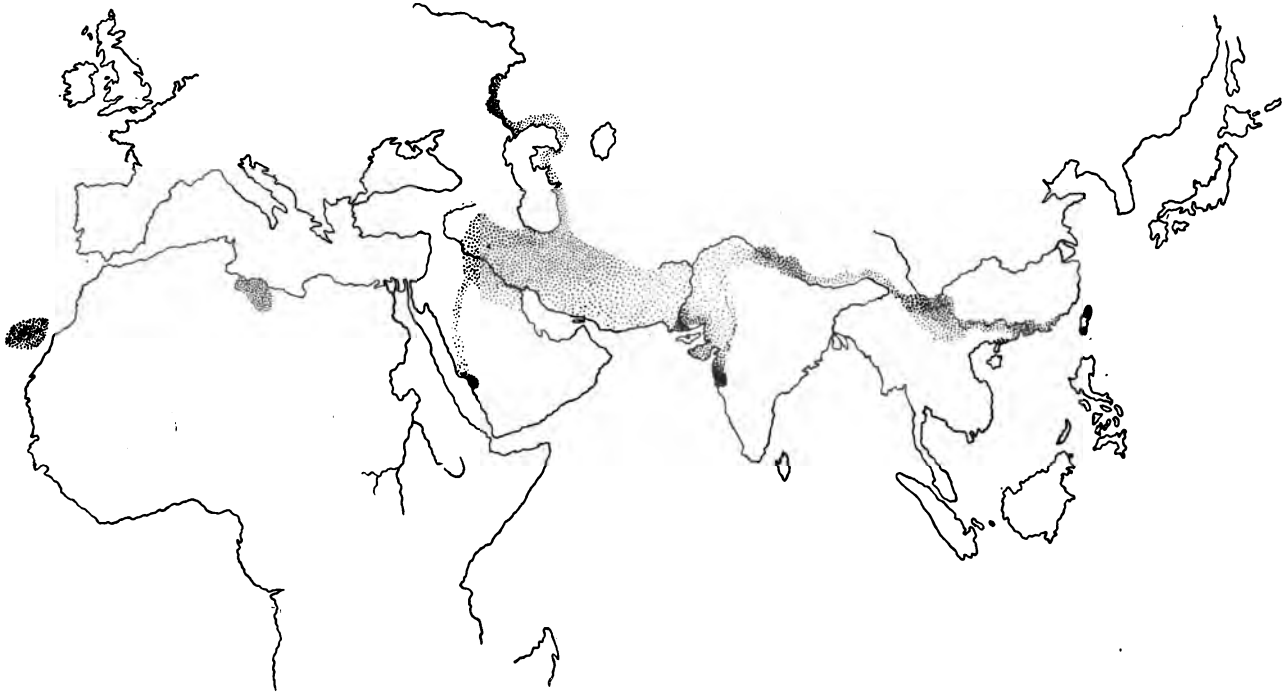
* Report of Dr. M. Wilm, dated Hong-Kong, May 20th, 1896.

while its *eastern* limit is the Island of Formosa, off the coast of China, where it is now prevalent. The *southern* limit is just beyond the Tropic of Cancer, but the *northern* range is indefinite. Since 1850 plague has never travelled further north than Astrakan, about 45° N.; but we have it on excellent and sustained authority that Moscow, Norway, and Sweden, and latitudes as far as 60° N., have within the present century been visited by plague. All limits are, therefore, fairly definite with the exception of the northerly limit. In other words, plague has been met with from 19° W. longitude to 121° E. longitude, and between 40° N. latitude to 19° S. latitude. Plague has never been known in the Western Hemisphere or anywhere south of 19° N. of the Equator.* The Basin of the Mediterranean and the strip of country which runs parallel to that sea across the Asian continent from Turkey to China may be roughly taken as its present belt during the nineteenth century, but the Mediterranean part of the belt has disappeared almost wholly within the present generation. A map of the plague-stricken districts within the last fifty years shows plague astride the Himalayas, with a giant limb reaching on one side the Red Sea and on the other the shores of the Pacific.

What determines the distribution it is impossible to say. Racial distinctions do not account for it, as the Mongoloid and Indo-European races are equally attacked. Neither ocean nor air currents have aught to do with its transit. It seems to thrive in the densely packed population of Canton and in the sparsely populated semi-desert regions of Rajputana and Arabia; the elevated plateau of Thibet and the low-lying valleys of the Euphrates are equally affected. It occurs during the summer's heat and the winter's snows. In fact, no known meteorological condition has any effect upon either the genesis or the exodus of plague.

As animals are affected, it is interesting to inquire what animals are affected, what is their geographical distribution, and what share, if any, they take in its spread. The animal which above all others is known to be affected by plague is the rat; but the wide geographical area throughout which this animal is met would seem to frustrate any attempt to determine a collateral distribution. On looking a little more closely, however, we find rats (the *Murinæ*) divided into two great sections—the *Mures*, inhabiting the Old

* This remark requires to be accepted with reservation, as it is reported that cases of plague have occurred as far south as Kolapore, in India, 16° 5′ N.



The dotted areas on the Map show Distribution of Plague during latter half of 19th century.

World (except Madagascar), and the *Sigmodontes*, inhabiting the New World (and Madagascar). Of the *Mures*, one sub-family, that of the *Nesokia*, is met with, reaching from Palestine to Formosa across the northern part of India. The southern limit in India seems to be where the great Bandicoot, or pig rat, exists, and here plague is unknown. This is the only animal which presents a habitation well-nigh corresponding to the present distribution of plague, and it is the animal above all others which is looked upon as being liable to be attacked by plague. The geographical distribution of this family of rats well-nigh coincides with the plague belt as delineated.

Pigeons are said to be severely stricken in Bombay at the present moment, but what kind of pigeon is attacked I have not yet been able to ascertain. When we have the information it will be interesting to note its geographical distribution.* We have no notice from China of pigeons being attacked by plague. The part the rat plays as a distributor of plague will be dealt with later, when we consider the relationship of animals to plague generally.

With regard to the continuity of plague outbreaks it would appear that in the plague belt the disease is ever active, now in one place and now in another. At times we find it in Arabia, then in Persia and the Himalayas, and the wave travels eastwards over Yunnan and Southern China to the sea. The disease oscillates, now east and now west, but with a fatal precision, making the slow swing of the pendulum of plague within a fairly definite area. A glance at the table giving the dates at which plague appeared in epidemic form since 1850 will confirm this. Taking the chief seats passed *en route* between the Red Sea and the Pacific, we find that plague appeared as follows:—In 1850, India; 1850, China; 1853, Arabia; 1853, India; 1856, Tripoli; 1858, Mesopotamia; 1859, Tripoli; 1863, Persia; 1867, Mesopotamia; 1869, India; 1871, Yunnan; 1872, Persia; 1873, Mesopotamia; 1874, Arabia; 1876, India; 1876, Mesopotamia; 1877, Tripoli; 1877, Russia (Caspian Sea); 1879, Arabia; 1880, Mesopotamia; 1881, Persia; 1881, China (Pakhoi); 1883, Afghanistan; 1884, Mesopotamia; 1886, Persia; 1887-89, Arabia; 1890, China (Yunnan); 1893, Tripoli; 1894, China (Canton, Hong-Kong); 1895, China (Macao); 1896, China (Hong-Kong); 1896, India (Bombay); and 1896, Merv (?). A study of this table shows

* A subsequent communication from Dr. Surveyor on the subject of pigeon infection makes it clear that plague bacilli were not found in pigeons.

the continuity of the course of plague, and how the eruption in one district is followed by its appearance in another. A slow traveller, it stays when it comes, and unless the scenes of its visitation are well garnished it becomes endemic.

The cause of plague is the advent of a definite bacillus, and it is the life-history of this bacillus which must in future occupy the attention of bacteriologists and clinical observers. The proximity of the bacillus is at the present day known only by its effect on human beings, and, it may be, rats as well. What fosters it appears to be bad sanitation; once established time alone will stay its ravages so far as any given epidemic is concerned, but there seems every hope that improved sanitation will check its recurrences.

The *prodromata* of plague may be divided into the remote and the immediate, but I will consider them together. Time and again it has happened that before an outbreak of plague, during a period varying from a few months to several years, buboes with fever have been observed in a more or less epidemic form. In 1877 the variety of plague which infected Astrakan, previously to the serious outbreak that occurred in 1878 some 150 miles higher up the Volga, was classed as a "pestis minor". No one died from the disease *per se* and but few people were confined to bed. Should such another outbreak take place in the neighbourhood the diagnosis will be readily cleared up by an examination of the blood, the tissues, and the excretion.

I was of the belief until within the last few days that this disease—this pestis minor—was of a nature wholly apart from true plague. From Calcutta, however, the news of the discovery of a bacillus allied in every respect to the Kitasato bacillus of true plague, and associated with the variety of plague known as pestis minor, has altered my views, although the bacillus is stated not to possess the toxic or virulent properties of the true plague bacillus, but to be similar in all other respects. The real question at issue at present is: Is there a disease of an epidemic character, attended by a low death-rate, characterised by polyadenitis and showing in the blood and tissues a bacillus resembling the bacillus met with in typical plague (malignant polyadenitis), and yet so apart clinically, that it is either a separate disease or caused by the same bacillus in a lesser or non-toxic form? In the discussion of this subject it is necessary to ask the following questions:—1. Does pestis minor occur as a precursor, as a collateral ailment, or a sequela of true plague? 2. Is it confined to the plague belt

delineated in the map? 3. Has it anything to do with plague? 4. Is it a disease *per se?* Dr. Payne, in the *Encyclopædia Britannica*, writes as follows: "In the minor form of the disease spontaneous swellings of the glands occur chiefly in the armpits, but also in the neck or other parts, which either undergo resolution or suppurate. There is a certain amount of fever; the temperature is rarely high, but has been known to be 104° F. The duration of the disease is from ten to twenty days usually, but may be eight weeks, for most of which time the general health is but little impaired and the patient able to go about as usual. It rarely, if ever, causes death, the only fatal case at Astrakan in 1877 having been so through a complication. The disease is not obviously contagious; whether it is propagated by infection or not is unknown. It is possibly rather of a miasmatic character. This form of disease has sometimes preceded or followed severe epidemics, as in Mesopotamia (Irak) on several occasions (1873-78) and in Astrakan (1877). Its importance in relation to the origin of plague has only lately been appreciated. It might be expected that gradations would be found connecting this form with the severe epidemic form; but this appears to be not usually the case, the latter form appearing somewhat suddenly and abruptly; hence the minor form has probably often been regarded as a 'distinct' disease, even when observed in plague countries." From all hands we have evidence of such a disease—namely, a pestis minor—benign polyadenitis. 1. The Russian outbreak at Astrakan in 1877 was probably, nay, certainly, of this nature, the low rate of mortality being sufficient to stamp it other than true plague. It is to be noted also that the typical plague which ranged in the same province appeared, not amongst the community afflicted with pestis minor, but in a district 150 miles away, where pestis minor was unknown. 2. In Calcutta, six years ago, a form of fever existed among the British troops there which in all points resembled the present form of bubonic fever which exists there now—namely, fever with specific glandular swellings. 3. We have it also from Calcutta that the natives are frequently afflicted with glandular enlargements during malarial fever. 4. In the far east, from Singapore, the Straits, and along the coast of China as far as Shanghai, between the years 1892 and 1896, we have accounts of a singular affection of the inguinal glands which occupied the attention of the Singapore and Hong-Kong branches of the British Medical Association. The correspondence was carried on between

the societies, and there is no doubt the affection at both places was identical in its nature.

I am compelled to draw attention to this subject in consequence of a paper which appeared in the *British Medical Journal* of Sept. 26th, 1896, by Surgeon C. C. Godding, R.N. Surgeon Godding refers to many cases of chronic glandular swelling attended by fever and anæmia, and he gives a careful clinical account of several such cases as having occurred in Hong-Kong. This is the first notice to the profession generally of a type of ailment with which all practitioners in the Far East are but too familiar. When the literature of the present outbreaks of plague comes to be written, the recorder will no doubt light upon these cases of glandular swellings given by Surgeon Godding, and take them as evidence of the association of pestis minor with the malignant type. We must, therefore, deal with them, come to what conclusions we may. When I brought the matter before the Hong-Kong Medical Society in 1893, I mentioned that I employed the term "bubon d'emblée" to designate the condition, and that I had treated thirty-eight cases in thirty-two months. My account of the ailment was as follows: "A slowly developing non-venereal bubo occurring in one or other groin, attended by general weakness, anæmia, and fever. The groin glands affected are discrete at first, but gradually the swellings amalgamate and a large mass as big as half an orange or larger presents. At the end of about twenty days points of boggy softness occur, and if the bubo be left to itself the skin becomes undermined and the pus finds its way to the surface through two or three or more fistulous openings. The swelling, when cut into early, shows a peri-glandular serous infiltration, and when pus occurs the gland will be found lying detached except by threads of tissue at one or two points. The gland when cut into shows many focal points of suppuration and breaks down quite easily beneath the fingers." Surgeon Godding advocates arsenic as almost a specific, but my own treatment latterly was early incision. It is necessary to make a clean sweep of every gland that can be felt or seen, as there is a great tendency to recurrence in any gland or piece of a gland that is left. Seeing that glands, except strumous glands, are seldom excised for other diseases, I examined the records of the Government Civil Hospital in Hong-Kong in connection with the excision of glands, and I found the following:—In 1891, nineteen buboes were scraped or excised; cervical glands, one. In 1892, I have not the record. In 1893, twenty-

seven buboes were scraped or excised. In 1894, twelve buboes were scraped or excised. 1894 was the plague year, and the list of operations is stated to be incomplete. Taking this evidence in conjunction with Surgeon Godding's and the private practitioners' in Hong-Kong and Singapore, we have weighty evidence of a widespread affection, be the cause what it may.*

Another form of glandular idiopathic enlargement is seen in what may be styled an epidemic form in children. In the year 1891, I reported twenty-three cases at the Medical Society in Hong-Kong, and since that date cases have frequently occurred. The affection consists of an enlargement of one gland, seldom more, in the necks of children over the sterno-mastoid, about the middle of its length. The swelling is seen *upon* the sterno-mastoid, but it might be found to commence, were it met with early enough, in a gland on its anterior border. The disease appears infectious and is attended by feverishness. There is no throat affection, nor is the seat of the disease in the parotid. We, the medical practitioners, styled the condition for "convenience" sake mumps, but we all admitted that neither the parotid nor the submaxillary glands were the seat of trouble. It will be remembered that the term "peculiar form of mumps" was employed by some to designate the Astrakan disease in 1877. 6. In the years 1893-4 and since, in Hong-Kong, cases of fever of a typho-malarial type occasionally presented in the third or fourth week a general enlargement of lymphatic glands, which lasted for a week or ten days and then subsided. They were deemed interesting cases at the time, but with the fresh light thrown upon them by recent investigation, more especially the researches in Calcutta, they assume a new aspect.

To sum up the evidence before us it would seem—(1) that a fever with glandular enlargement, pestis minor, a "benign polyadenitis," is an established disease; (2) that it may be independent of plague or malignant polyadenitis; (3) that the two may coexist; and (4) that the one may exist independently of the other.

As to *infection* and *contagion*, direct contagion, that is, transference of the disease from the sick to the healthy by bodily contact, would appear at the first glance to be an element in the transmission of plague. How otherwise can we explain the circumstance that, when a case of

* Europeans were mostly attacked by these buboes, being 88 per cent. of the cases operated upon.

plague occurs in a house, those dwelling in the house, and brought in contact with the sufferer, are almost certain to be seized, and not only so, but few if any of the other inmates escape? The next in order to be seized by plague are the relatives of the sick and the visitors to the house, who, going to their own homes, set up a fresh focus of infection. Of the community, medical men, clergymen, and attendants, who are brought in contact with the sufferers, more readily acquire the disease than others who are alien to the family and house. These facts would appear to prove conclusively the contagious nature of plague. But there is another side to the argument. As long ago as 1835, when plague was epidemic in Egypt, out of ten French medical men who attended the sick only one contracted the disease. Further, we have the well-known case of Bulard during the same outbreak, who, anxious to prove his contention that the contagious nature of plague was much exaggerated, for two days wore the shirt of a patient who had died from plague. Yet Bulard did not contract the disease. More recently we have the evidence of the Hong-Kong epidemic in 1894. None of the European medical men in attendance on the plague patients, in all some fifteen in number, were attacked by plague. Those men were freely exposed in the wards and at post-mortem examinations, yet none were infected. Further, none of the British or Italian born nurses—these were the only two European nationalies represented—were attacked by plague during 1894. But a more interesting fact still is that none of the Chinese students of the College of Medicine were attacked. For six weeks these men were on duty in turns night and day in the Plague Hospital. Eight of them acted as ward attendants and clerks throughout the period and were consequently in constant contact with the sick. A racial immunity might be claimed for Europeans, but in face of this fact it cannot hold good. If there be any truth in the acquisition of disease during an epidemic by fear or nervousness the case of these students refutes the legend. They saw that the Europeans escaped and how terribly their countrymen suffered; they had to contend with the superstitious beliefs of their country, inherited and acquired; they were breathing the same air, eating similar food, hearing daily, hourly even, of the deaths of their friends, relatives and acquaintances, yet none of them succumbed to plague. We have, therefore, two opposite conditions before us—namely, plague entering a house and seizing practically all the

inmates; and, on the other hand, medical men, nurses, and native students acting as clerks and dressers escaping completely. Where, then, lies the truth of the contagious nature of plague?

As usual, there is a channel of escape from either of these two extremes of argument. The immunity above related does not extend indefinitely. The Europeans attacked during the 1894 epidemic were chiefly soldiers of the Shropshire Regiment, then stationed in Hong-Kong. When plague required sanitary interference beyond the power of the local board, these soldiers came forward and volunteered to help in the work of cleansing and purifying the city. The work assigned to them was of the most laborious order, and they were exposed to a concentration of the contagion, for the field of their duty lay amongst the dwellings of the poorest class of Chinese who were suffering from plague. Those houses were of the vilest kind of habitation mankind is acquainted with; small, windowless, low-ceilinged, reeking with filth and excretions, they presented infection in its most concentrated form. Further, the soldiers had to use spade and shovel to dig down into the accumulated layers of dirt on the floors of the houses and throw it into the street. It was this occupation more than any other to which is attributed the infection of the soldiers. During the 1894 epidemic two of the sisters of the Italian Convent contracted the disease and died, but they were neither of them Italian-born; they were both Eurasians and in very humble circumstances. In 1894, also, three Japanese medical men contracted plague, of whom one died. The two who recovered were both considered to have inoculated themselves at a post-mortem examination on a case of plague. The facts recorded would seem to show that, during the 1894 epidemic in Hong-Kong, English practitioners and nurses enjoyed an immunity which did not extend to the Japanese medical men or to the nurses of Eurasian blood.

The evidence of the subsequent outbreak in Hong-Kong in 1896 goes to show that Europeans were much more frequently attacked than in 1894, and two of the nurses in attendance on the sick contracted plague. One of the nurses was a British-born nursing sister at the Plague Hospital, where she had been for many months. She never was ill until the very end of the epidemic, when she got a sharp attack of plague, from which, however, she fortunately recovered. The sister (Katherine M'Intosh) believes she was infected by a little child she nursed, and

whom she frequently carried about in her arms. The other European nurse was an Italian-born sister in the convent, who believed she was infected in the same way—viz., by nursing and carrying about a child sick from plague. The Italian sister died from her disease. It is interesting to note that the patient from whom the English sister contracted her disease recovered, whereas the patient of the Italian sister died. Was the recovery of the English sister due to a lessened degree of infection, seeing that her patient recovered, and the death of the latter due to the extreme malignancy of the emanations from the body of the dying child? is a pertinent question. Nor is this all, for yet another sister in the convent died, presumably infected whilst nursing her companion. These three deaths seem to point markedly to contagion rather than infection as the means of spread, for although there were some 600 persons in the convent, none of the other inmates were afflicted thereby.

Gathering this evidence together and trying to focus it, we find that the soldiers who were affected were exposed—if there is any truth in intensity of contagium at all—to the most concentrated form of the poison. They worked not in the streets in the open air, but in reeking hovels for several hours together. Intimate and prolonged contact with the contagium in an active condition seems to be the explanation of their infection. The same is to be said of the two European nurses who were seized. Both attributed their ailment to intimate and prolonged contact with a generating source—viz., a child carried in the arms not for a few minutes, but for several hours daily extending over several days. That there are, moreover, degrees of power in the contagium, even when emanating from the human body, seems to be supported by the nurse and infecting child both recovering in the one instance, and the nurse and infecting child both dying in another. Bulard sleeping in the dead man's shirt proves nothing further than that the plague-infected garment did not generate poison of an intensity sufficient to infect. The poison grew every moment more dilute, but a nurse carrying a child, throwing off contagium continuously, is an exposure of a different stamp. The immunity of the medical men and Chinese students does not annul the effect of prolonged and intimate contact as being a means of spread; for it must be remembered that the medical man "visits" only, he does not stay for a day beside one case as the relative does, nor does he sleep or eat under the same roof, as does

a nurse. In this way also the immunity of the Chinese students is to be accounted for. They performed their task in a newly-built, commodious, and roomy mat-shed. Freely ventilated by being raised three feet from the ground, and its lofty roof constructed so that air swept across between the side walls and the roof with large apertures in the roof itself, the chance of contracting the disease from so diluted a form was reduced to a minimum

The practical lesson to be learned from these statements is that free ventilation, and attention to ordinary hygienic rules, lessen the chance of contracting the disease to a very great extent, not only for Europeans, but also for natives; but that all are liable to contract the disease when either from the calls of duty or wilful neglect exposure is extreme.

What has been termed the "miasmatic" method of infection bears distinctly upon the maintenance and epidemicity of plague. Miasmatic infection implies a soil-bred disease—a disease existing, as we assume malaria does, in the earth itself, in the water, or in some particular form of fermenting or decomposing material in which the germ finds a nidus. In a neighbourhood so infected the disease is endemic, as manifested by the fact that again and again the disease recurs—that after a few months of seemingly complete disappearance the disease crops up and runs a more or less severe course. The time of its disappearance may be merely one of hibernation, or the climatic conditions may be unsuited to the activity or the evolution of the bacillus of the disease; but when a sufficiency of time elapses, or the requisite quantity of heat, moisture, and whatever else is necessary to the growth of the bacilli are attainable, the revival of its infective power is speedily manifested. Whilst comparing malaria and plague as miasmatic disease it is not to be inferred that there is any bearing in the relation of the one to the other. In fact, Dr. Mouchard, writing from Yunnan, cites the cases of Mentz, a town on the hills, and Manhao, a town on the river, 3,600 feet lower. In the former plague is endemic, but in Manhao malaria is so great a scourge that the Mentz muleteers refuse to spend the nights in the town.

Infection of, and by, Animals.—In support of plague being a soil-bred—a miasmatic—disease is the fact, so often announced, that rats are invariably affected. From Canton, Hong-Kong, Macao, Pakhio, Yunnan, from hills in the North-West Provinces of India, and, most recently of all, from Bombay, the observation is constant, definite,

and most pointed. The first time particular notice is taken of the death of rats in connection with plague is in Mr. Rocher's report on the Yunnan plague, some twenty years ago. The Chinese firmly believe the poison emanates from the soil; so far is their conviction carried that they estimate with mathematical precision its gradual rise above the ground. The graduation is estimated by the successive animals that are attacked. The sequence is affected apparently by the height of the animal's breathing apparatus from the ground, the small animals being the first seized and the larger or more elevated animals the last to be attacked. Rats, poultry, pigs, dogs, goats, sheep, cows, buffaloes, and, last of all, man as being the tallest of these, is their scale of infection. This is not to be taken seriously, but the infection of some of these animals is undoubted.

Rats.—On all hands rats are reputed to behave peculiarly and with a wonderful constancy. Before, or it may be during, an epidemic of plague, or before the individuals in any particular house in an infected locality are stricken, the rats leave their haunts and seek the interior of the house. They seem careless of the presence of man, and run about in a dazed way with a peculiar limping jerk or spasm of their hind legs. They are frequently found on the bedroom floor, or on the tables, but not unfrequently their death is known by the putrefactive odour of their decomposition arising from beneath the flooring. So pertinent has this rat affection become, that during the epidemic in Macao in 1895, the Chinese and Portuguese left their houses when the diseased rats invaded their premises. The cause of the rats' behaviour is undoubted disease, and the symptoms tally wonderfully with plague symptoms of man. Dr. Rennie examined them carefully in Canton and noted the following post-mortem appearances: (1) the stomach was distended and filled with particles of food, sand, and indigestible substances, and the mucous membrane was red and inflamed towards the pyloric end; (2) the liver was much enlarged and congested, and contained ova of tænia and distoma; (3) there was congestion at the base of the lung present in about 40 per cent.; and (4) glandular enlargement was present in 30 per cent. of those examined. There is no doubt now that the disease in the rat and man are identical. The bacillus of plague has been met with in every case of rat disease of this description where it has been searched for. The infection of the rat is raised from being a mere popular belief into one of scientific precision,

and we must now accept the rat at any rate as one animal liable to plague. Whether the rat is affected previously to, coincidently with, or subsequently to, man being attacked is open to question. Is the disease amongst rats a forerunner of its outbreak in man, and if so, are they a means of infection? These are, of course, two separate questions.

Surgeon-Major S. J. Thomson, Deputy Sanitary Commissioner, North-West Provinces, India, who made excellent reports on the outbreaks in Ghurwal and Kumaon in 1886 and again in 1888, writes as follows*: "A fact mentioned in the Hong-Kong epidemic by Dr. Cantlie is the great mortality amongst rats. This is also most marked in the case of outbreaks of *matra-mari*, a hill plague; but I should add that it is generally observed *before* the appearance of the disease amongst human beings. So well is this recognised by the people that they will sometimes vacate their villages for fear of an outbreak of matra-mari when once this rat mortality has become distinctly noticeable." In Canton, Dr. Rennie writes that "from districts of the city where the diseases have lasted for some time rats entirely disappeared; but in other parts to which plague subsequently extended the rat mortality was most marked. So convinced were the Chinese of the power of the rat to infect that certain officials took steps to have all the dead rats collected, and at one gate as many as 22,000 rats were collected by May 8th, which, taking into consideration the date of the outbreak, must imply that the collection was made in a few weeks." The *Times of India*, Sept. 30th, 1896, contains the following:—"It was known *more than a month ago* to all the people of Mandvi (the district in Bombay where plague first broke out in September, 1896) and to all the municipal sweepers in the district, that the rats were dying in thousands all over the districts. They were found dead and dying almost everywhere and in places where dead rats were never found before. The children amused themselves every morning by throwing them from the staircases into the streets and gulleys. The great rat mortality only became known recently, and yet what a volume of information was it capable of conveying if it was only rightly used. It is universal history that rat and plague mortality go together; the merest tyro in hygiene knows the fact," etc.

The rat therefore seems to be infected before the human

* *Brit. Med. Journ.*, Sept. 8th, 1894.

being; and the fact of dead rats being found about the house during the plague epidemic is a true warning that the inmates of the house will in all probability be affected. How is the rat infected? 1. Experiment proves (*a*) that the rat is inoculable and dies from a disease identical with bubonic plague in man; the inoculation of both the blood and inflammatory fluids of the plague-infected is effective; and further, a pure cultivation injected hypodermically also sets up signs and symptoms of rat plague and causes death within a few days; and (*b*) that supplying the animal with plague-infected food, whether flesh or cultivated germs, causes plague in rats. How does the rat in his natural state become infected; by inoculation? We cannot assume that every rat has a scratch or open wound by which it is infected. It is more likely he inhales or imbibes the poison either as a miasm in concentrated form or he ingests it with his food. It may be both forms of infection are at work; and, further, it must not be forgotten that rats are cannibal in their habits, and direct contagion must play a large part, if not the major part, in the general infection of their tribe. That it is some general infection and not a chance inoculation is manifested by their sudden and simultaneous seizure—a seizure so sudden that scores of dead and diseased rats will be found in a single house of any size within a few hours. An interesting observation in connection with the Calcutta affection is "that although Calcutta was believed to be the seat of plague, and the plague bacillus was pronounced to have been proved in six cases, no rats died." This was regarded as a peculiar and shall I say, an unprecedented condition, but the explanation is simple enough. The rats did not die, as true plague did not exist in Calcutta, the cases of fever with bubonic swellings being found to be minus a toxic bacillus. This is in accordance with all previous experience. The Calcutta form of disease is benign and no record of rats dying during its prevalence has ever been made. Are we to doubt our belief in the diagnostic value of the bacillus or admit a new disease into our category—a benign polyadenitis? It may be we are called upon to do neither, for we have evidence to show that non-virulent forms of the bacillus can be cultivated and that a bacillus can be found in the soil and cultivated which behaves like a non-virulent bacillus extracted from true plague cultures. These points will be discussed later. 2. An observation by Dr. Thomson in the North-West Provinces of India is worth recording. "In these hill tracts the huts are usually double storeyed,

the upper storey being occupied by human beings, and the lower by cattle, goats, etc. It is the custom of the villagers to store the grain in pits under the floors of the huts, and the soil is very foul from the excrement of animals, etc. The theory among the people is that the rats eat the grain, which is often musty. Most of these hill people are very poor, and would consume such grain, which is their principal food, even when musty and clearly affected with fungoid disease." It is some widespread source of infection by which the rat is infected. Dr. Rennie tells us that the places from which the exodus takes place are left without a rat to be seen.

If not from grain or from inoculation, whence comes the poison? From the soil? Dr. Rennie found sand and indigestible material in the contents of the dead rats' stomachs. May this sand have been the source of infection? I kept guinea-pigs on soil from the grave of a plague patient who had been dead for two months; the guinea-pigs were not infected, but perhaps the earth had hardly had time to become infected. If the poison is miasmatic and emanates from the soil as a gas or cloud the rat in his close stifling quarters is exposed to a concentrated inhalation. I have already mentioned the soldiers employed in digging out the filth from plague dwellings being seized, and the infection was presumed to be brought about by the intensity of the virus. Therefore, we cannot presume that the rat can be infected by a miasm merely. Of the three forms of infection discussed we may safely lay aside inoculation. We may hesitate over the infection by fungoid grain, but although the musty grain would render the disease endemic it could not account for an epidemic spreading to districts where the grain is sound, unless we assume that the rat is the carrier. We are left, therefore, either with actual consumption of poisoned material or the concentrated emanation from an infected soil as the means of introduction into the rat's economy.

Snakes.—Dr. Thomson remarks*: "Moreover, when rats are thus dying dead snakes also are said to be found about the site in unusual numbers." Later he remarks: "The snakes eat the rats." From the North-Western Provinces of India we also have a record of snakes being affected during plague outbreaks. The snake is not observed as one of the animals affected in the list given by the Chinese, but the infection is natural, and the infection through their consumption of the rat is feasible. The

* *Brit. Med. Journ.*, Sept. 8th, 1895, p. 560.

possibility, however, of direct miasmatic infection must not be lost sight of.

Dogs.—In the *Chinese Customs Medical Gazette*, in the forty-seventh and forty-eighth issues, we read that dogs died during an epidemic of plague. The observer ascribes it to the dogs eating the flesh of human beings or any other animal dead from plague. In the same paper it is described that in Yunnan the dying are removed out of the houses to repose in the streets in accordance with Chinese customs, and that those dead and dying from plague are found outside the dwellings or even outside the town walls. He has, moreover, seen the dogs eating the flesh of those dead from plague. In Hong-Kong we had no special mortality amongst dogs, nor have Canton or Macao anything to say on the subject, but the dead were better cared for than in Yunnan and no dog could get at them. It would seem as though the dog were infected by eating the flesh. In the places mentioned such an opportunity was not afforded him, but in the town of Mentz, Yunnan, the custom as to the disposal of the dying seems to explain the infection of the dog.

Pigs.—It is even more important to ascertain whether or no the pig is infected. Pork is largely consumed by Chinese; and, moreover, Europeans in the East seldom or never eat Chinese fed pork. The Chinese died from plague, whilst the Europeans enjoyed an immunity from the disease; nothing seems more conclusive than that pork was the infecting food. Moreover, most of the pork brought to the Hong-Kong market comes from Pakhoi, a town where plague has been endemic for years. The first point to be settled is—Does the pig suffer? Not as a rule. The pigs from Pakhoi showed no falling off in numbers, and they were not sickly; the former condition would have obtained had plague thinned their numbers in Pakhoi, and the latter would have been found out when they reached Hong-Kong, where careful inspection of all food supplies is made. Dr. Mouchaud writes that the pigs died in Yunnan, and he states that "we saw on some roads dogs and pigs feeding undisturbed on corpses which no one cared to bury. These animals fell victims to their greed and succumbed to the scourge." Further, plague has been produced in the pig experimentally, all of which goes to prove that pigs die from plague after consumption of corpses dead from plague and inoculation; but that pigs are not primarily liable to plague in the same way as rats and pigeons seems conclusively shown.

Oxen, sheep, and *goats* are not known to be subject to a disease allied to plague, nor are they affected by any fatal malady during the time plague is epidemic. As regards sheep, Custaldi states that plague in Kurdistan was preceded by a fatal epizootic among sheep; that they had plague, however, is not stated.

Jackals are known to be subject to the disease in Yunnan. They are more prone than either dogs or pigs to consume exposed human bodies, and are likely animals to be affected.

Pigeons.—Dr. Surveyor, in a private letter to me dated Nov. 13th, 1896, Bombay, asks: "Did you notice any mortality amongst *pigeons* from this disease?"* In reply I have to state that I did not, nor did any observer in China, as far as I know, draw attention to the infection of pigeons. If stored grain is the source of infection it is easy to imagine that pigeons would be perhaps one of the most likely species of birds to be affected; and it but draws attention to the disease of grain still more pointedly than before. Several observers in India are most decided in their statements as to the grain source of the disease, and that the rat and pigeon are the sufferers seems to point somewhat in that direction. On the other hand, it must be remembered that the ordinary barn-yard fowls are not (except in the London Plague of 1111 A.D., and it is so long ago that it may be discounted), so far as we know, struck down by any disease resembling plague; so that, although the grain origin of the disease may be worth investigating, we cannot turn towards it with any *a priori* hopes of elucidation. It will be seen at once that the infection of birds will render the possibility of the rapid spread of plague on its appearance in isolated districts easily explainable. It is not comfortable news that we have a winged distributor of plague.

To sum up our knowledge, we find that pigs, dogs, rats, snakes, jackals, and pigeons(?) are affected by a fatal malady whilst plague is epidemic amongst human beings. Pigs and dogs are believed to be attacked only after consumption of plague flesh. Snakes, by the swallowing of rats, and possibly jackals, are infected in the same way. None of the purely herbivorous animals—horses, donkeys, oxen, sheep, goats or rabbits—are attacked. It is the flesh-eating animals which are the sufferers. Of course, the rabbit, guinea-pig, and a number of animals can be inoculated experimentally or made to consume plague-diseased food,

* Dr. Surveyor states, subsequently, that for a time only was this a popular belief.

but no herbivorous animal acquires it by the natural process of infection. If it is by consumption of flesh that animals are infected, how do human beings get it? The animals affected seem to become infected from flesh: the snake from the rat, the jackal, pig, and dog from eating the flesh of persons dead from plague; but how does the rat become infected? Could we find that out, we should very likely know how man is infected. One animal missing in the scale is the cat: if rats perish from plague surely their mortal enemy, the cat, would be diseased. Perhaps the instinct of the cat teaches it to avoid the diseased rat. [In Hong-Kong during 1894 many cats died in a peculiar way—the chief symptom being paralysis of the hind legs.]

In the discussion about the rat infection it must never be forgotten that, if we take it for granted that the disease attacks rats before human beings become victims, we must consider the question, "Has the rat ailment to do directly with the subsequent outbreak in man?" It is very difficult to see how the rat could affect man except by *infection;* and one may hesitate to believe, from what we know of the concentration of poison necessary to infect, that such is possible. It might be that the diseased rat, by *treading,* or dropping upon, or biting the food of man, would thereby poison the food. It will at once occur to one that sick rats, like sick men, would not eat. This we have no proof of; in fact, it is the other way, for Dr. Rennie states that in his post-mortem examination on rats in Canton he found the stomachs pretty full of food, and rats during incubation and first days of illness from experiment eat fairly well. What we may conclude from the study of the infection of animals is: (1) that the rat is the animal most liable to be attacked by plague; (2) that rats suffering from or dead from plague may infect other animals, such as snakes and jackals, who consume them; (3) that rats are always affected by a disease similar to plague at the same time man suffers; (4) that the rat may infect man, but the means of conveying the contagium is not known; and (5) that during the prevalence of benign plague (pestis minor), rats do not die.

Incubation.—The time intervening between exposure and the development of plague has been carefully observed and noted throughout many epidemics of this century. The conclusions arrived at by all who have written an account of their Hong-Kong experience gives from three to six days as the period of incubation. Dr. Lowson makes it seldom under three or over six. Professor Aoyama gives

it at from two to seven days. If by that statement he means from the end of the second until the beginning of the seventh day, his recorded observations agree exactly with Dr. Lowson's and others. Only one case seems to break the record: it is a case referred to by both Dr. Lowson and myself. A Chinaman in the gaol at Hong-Kong developed plague after being nine days under lock and key. From June 11th until June 20th he was apparently well, but on the evening of the 20th he became feverish and developed plague. This case gives nine days as the apparent period of incubation, and may be taken as the limit in that direction. Dr. Lowson states, however, that the man complained of being ill for two days before being placed on the sick list, thus reducing the period to the seventh day; and although the man was put in the cells on the 11th, it was not until the 12th that he was really beyond contact with the outer world. Further, during an epidemic of any kind, isolated cases occurring here and there in the infected centre, where contagion seems difficult to explain, must not be taken as sufficient to seriously arrest attention even when a gaol wall intervenes. An infected area is one in which any dweller may succumb to the disease, despite all precautions of isolation. Whilst mentioning this case, I do not think we have evidence to place the period of incubation of malignant polyadenitis beyond the end of the sixth day after exposure. Be it observed, however, this extends only to plague infection during an epidemic.

There is a notification of two or three cases of plague as having occurred at the Seamen's Hospital, Greenwich.* The following is a statement of the facts: "About the middle of September a man, a Portuguese-Indian steward, was taken into the Branch Seamen's Hospital and died there very suddenly. He was only 'in' about forty-eight hours, and no suspicion of plague arose. On the last day in October there was reason to consider that another patient at that time in the hospital was suffering from plague; the previous case was thought of, and it was agreed that if one was plague, the other in all probability was of the same nature. The man died; but the medical authorities were not in entire agreement. As a precautionary measure, however, the body was buried in a leaden coffin. The matter was referred for bacteriological examination, the glands of the body were examined, and a bacillus, which

* *Brit. Med. Journ.*, December 12th, 1896.

presented the known characters of the plague bacillus, was found." In connection with this subject there are one or two points calling for comment. 1. The dates given (if correct) would show the vessel, in which the infected persons were, left Bombay about the end of August. Now, we have no direct medical evidence that plague existed in Bombay during August. The earliest official intimation of plague in Bombay was when Dr. Viegas reported, at a meeting of the Sanitary Committee on Sept. 23rd, 1896, that the disease had prevailed in the locality for a fortnight, thereby assigning Sept. 9th as the date of commencement. The first case, therefore, at the Greenwich Hospital could apparently not have been infected from that source, as the vessel could not have been less than seventeen days on the way. Either it is not plague we are dealing with at Greenwich (I firmly believe it is), or the infection must have come from some other source than Bombay; or yet a third condition may obtain, which heretofore we recked not of—namely, an indefinite period of incubation, during which period "benign" plague had become malignant. This subject will be discussed later.

In elucidation of these cases and the source of their infection, I think I can throw some light. I stated above that there was no official evidence of the presence of plague in Bombay before Sept. 9th, 1896—that is, ten days after the first Greenwich Hospital patient sailed from Bombay. This may not be quite correct, for I find in the *Times of India*, Sept. 30th, 1896, the following statement:—

"In Bombay for two months certainly, in all probability longer, the native inhabitants of the neighbourhood of Maudvie, where plague broke out in Bombay, seemed to have been alive to the fact of the existence of a malady resembling plague before the malignant type developed. They had organised processions in August to propitiate the goddess of Plague. This either proves that true plague was present long before the authorities knew of its existence, or that a disease resembling plague was mistaken for the malignant type." According to this evidence, plague may have existed in Bombay in July, 1896. This evidence will, I hope, help those who are dealing with the Greenwich Hospital cases to disentangle the incubation period difficulty. The second case reported from the Greenwich Hospital had been on shore fourteen days before falling sick. Therefore, allowing seventeen days for the voyage, thirty-one days must have intervened between exposure in Bombay and the illness.

The occurrence of several cases of pestis minor in Calcutta, in which the bacillus was found, indicates a more cautious acceptance of diagnosis resting solely on bacillary evidence than we have heretofore contemplated. The first case dealt with and reported upon in Calcutta gives an instructive history. The patient, a lad aged seventeen years, came from Bombay, where evidently he had been exposed to infection, as his sister, who accompanied him, had seen several cases of plague in Bombay. Fifteen days before leaving Bombay, he had noticed swellings first in one groin and then in another, but never felt ill until his arrival in Calcutta on Sept. 24th. He was seen and carefully examined in Calcutta by honest observers, and a diplobacterium identical with the Kitasato bacillus was found in his blood. Not only so, but the clinical symptoms of plague were most manifest. It would, therefore, seem from these two well-authenticated cases that glandular swellings may attack a dweller in a district where true plague rages, and that at a subsequent period (a period which in the Calcutta patient was not less than twenty days and in the Greenwich Hospital case could not be under seventeen days) may become virulent and kill the patient by a true plague; in fact, that a pestis ambulans may become a malignant polyadenitis in the same person, even although the patient is removed from the infected zone. This may be true, although it is not at all a comfortable doctrine; but I can come to no other conclusion having regard to the evidence before me. The incubation period of plague, therefore, may be fixed as from three to six days whilst an epidemic rages, but the evidence to hand within the last few weeks, from Calcutta and from London, renders the period of incubation of ambulatory plague uncertain.

Invasion.—The onset of the disease in malignant polyadenitis is known to be usually sudden and severe. The patient is seized with headache, fever, and prostration, and compelled straightway to seek his bed. In a few hours or a few days a swollen gland developes, and the disease runs its course. But we have on record many cases where high fever existed and the patient did not even feel ill, or where a gland swells up with no general feverish state. Dr. Lowson, in his valuable report, states: "In one case I took the temperature of an Indian who *looked* ill, but who had merely come to see about the burial of a friend, and who complained of no unpleasant symptom. He was rather amused at my using the thermometer, but I found he had a temperature of 103° F., and at the same time dis-

covered a small cervical bubo." Be it observed, as bearing upon the intensity of the virus, that this was a non-fatal attack of plague. In many cases a bubo may precede the fever, the patient suffering no inconvenience even. Such a case is that recorded from Calcutta, where "the patient, a boy from Bombay, had had from fifteen or twenty days before developing fever a swelling in either groin. Subsequently he developed the ordinary symptoms of plague, and the diplococcus was found in his blood." These two cases are sufficient to show that fever or plague may exist and the patient not be aware of it, and that glands may be affected for a fortnight before the disease declares itself in the usual way by headache, fever, and prostration. The onset of the disease, therefore, even during a plague epidemic, may not be so definite as we should like to believe, and as yet we have no charts recording the condition of the patient between the time of exposure and the invasion.

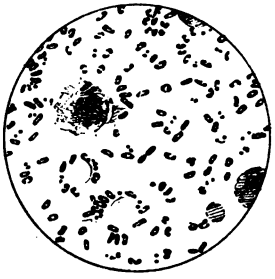
Varieties.—It is not my intention to mention merely the varieties of symptoms which predominate at any one time or throughout any one epidemic, but to draw attention to varieties which are so pronounced in their character as to justify the belief that, whilst dealing with this subject, we are actually attempting to bring in line under one heading diseases which may be even separate species. The commonly accepted types are these: (1) abortive (or larval) plague; (2) typical plague; and (3) fulminant plague. The two latter are so well understood, and have been so largely and fully dealt with elsewhere, that there is no advantage in recapitulating what can be read in any text-book; but I wish to draw attention to the first-named. The abortive form is sometimes styled larval, ambulatory, or pestis minor. The names partly declare the character of the disease, and create a condition so apart that it has at all times been separately described. It is at the same time so indefinite in its duration, so unstable in its relation to malignant plague, and so uncertain in its onset and departure, that up to the present no scientific precision has been assigned to the relationships. As long ago as the year 1666, when plague ravaged London, we have it on the authority of Sydenham, that "at times tumours may break out without either fever or any important symptoms. . . . Those to whom this happens may walk about in public as usual, and attend to the common duties of life. . . ." Foderè, writing of plague in 1820, observes: "In the Levant and in the Marseilles epidemics of 1820 cases were to be seen which were not

ushered in by any alarming symptoms, and where the natural functions were undisturbed, and where buboes and carbuncles appeared without fever or only with slight fever, or the buboes went on to a healthy suppuration more or less prompt, or even disappeared and went on to resolution without the help of art, without any inconvenience, and with a perfect integrity of all the functions. This state is comparable to benign small-pox, during which children play together and walk in the streets without any precautions, no care being taken of their treatment, and yet terminating favourably. It is the benign plague of authors, which is observed when the disease commences and when it is at its end, though it is rarely seen in the middle period, which is entirely devastating; *but it is not less plague, and it no less merits the attention of physicians and magistrates.*" Again, in all recent epidemics in Tripoli, Mesopotamia and Astrakan, non-fatal mild bubonic disease occurred both before and after the epidemic. We have it on the authority of Dr. Dickson that in Mesopotamia, previously to the manifestation of plague, glandular swellings free from fever prevailed. All this evidence seems to indicate a close connection between benign and malignant polyadentis; but we have some evidence of the separate existence of the two diseases. 1. In Mesopotamia Tholozan records: (*a*) that mild non-fatal bubonic plague existed from 1856 to 1865, during which period (nine years) no malignant type was observed; and (*b*) that after the epidemic of malignant plague (1866-67) passed over, the same form of mild plague existed. Did it exist during the epidemic? This is not known, for it is not likely that persons suffering from so mild an ailment would present themselves for treatment to foreign medical men at a time when all swellings were viewed with suspicion. There is, therefore, no evidence to show that the two diseases did not co-exist, seeing that it existed nine years before and perhaps as long afterwards. The present belief is that the mild develops into the malignant type, and when that subsides it is once more deprived of its malignancy and becomes benign. We have no evidence of any disease behaving in such a manner to such an extent as claimed for plague. 2. In Calcutta, six years ago, an outbreak of mild non-fatal plague occurred in the fort amongst the troops, but it was not followed by the malignant variety. 3. At the present moment in Calcutta there is reported to be a form of fever with glandular swellings, and also a form of glandular swellings without fever; and it is further stated

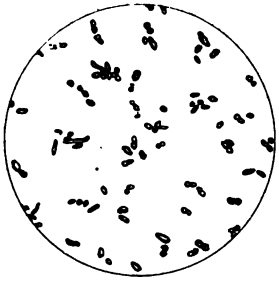
that these are constantly met with in medical practice in Calcutta. 4. In 1890 the variety of idiopathic buboes, of glandular swellings with fever, and of mumps met with in Hong-Kong, before referred to, was not followed until four years afterwards by plague. These records go to prove :— 1. That long periods of time may intervene between the prevalence of specific glandular swellings and the outbreak of malignant plague. 2. That glandular swellings may exist for nine years before malignant plague appears, and continue for years after it has disappeared. 3. That independent outbreaks of glandular swellings (pestis minor) may come and go, and no plague supervene.

The latest evidence we have from Calcutta is conflicting in the extreme. We have it to hand on trustworthy evidence that people are suffering from fever with glandular enlargement: that the disease is regarded as pestis ambulans—a mild bubonic plague—and that *the diplobacteria of plague are found in the blood.* We have hitherto regarded the presence of the bacillus in the blood as the one token of the disease being malignant in type; but we have now contrary evidence to consider, and if we are to accept it, we must believe that specific glandular swellings in the plague belt may be caused by a definite bacillus, and at the same time admit a varying degree of malignancy to that bacillus. Professor Cunningham—a most competent observer—declares that the bacillus found in the Calcutta cases is non-infective, and that probably it is not the same bacillus as that described by Kitasato in 1894. This is stated in the face of positive evidence by careful observers, which cannot be set aside thoughtlessly. Time alone will answer this question. In the meantime, however, it is feasible to suggest that there may be but one bacillus; and that according to its state of development, or in consequence of its evolution, different types of the disease may prevail in the human body, each associated with a definite train of symptoms, characterised in the one by glandular swellings merely, and in the other by a toxic infection of extreme intensity.

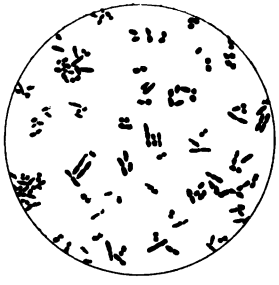
The bacillus.—The bacteriology of plague began with the advent of Kitasato from Tokio during the Hong-Kong epidemic of 1894. On June 14th, 1894, Kitasato demonstrated the bacillus, which was confirmed afterwards by Dr. Yersin, of the Pasteur Institute in Saigon, working in an independent manner. The bacillus has been variously described as (1) a diplococcus; (2) diplococcus enclosed in a delicate capsule; and (3) a short bacillus with the ends



Plague Bacilli from Spleen of Mouse.
(*Yersin, Hong Kong*, 1894.)



Plague Bacilli Cultivation.
(*Haffkeine, Bombay*, 1896.)



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(*Surveyor, Bombay*, 1896.

somewhat rounded, and a clear space or band in the centre. Professor Woodhead writes :—" The size varies considerably according to the staining method employed, the organism appearing rather smaller in the watery gentian violet solution than in the glycerine fuchsin solution. It is plain that the size of the capsule and its staining capacity vary very considerably, and it is quite possible that this appearance of a capsule is like that which is sometimes met with around the bacillus of anthrax stained in special solutions, in that it may be simply a refraction from the albumen from the constricted bacillus due to the drying and other processes of preparation." In the figures drawn by Woodhead, an organism was met with in some specimens which seemed to indicate a development after death. The last statement may be the most important of all, and its confirmation will be anxiously looked for during the present outbreak in India. It may be the first step towards a knowledge of the life-history of the bacillus, in the same manner as the flagella of the plasmodium malaria are found to develop in the field of the microscope some half hour after removal from the body. Dr. Yersin undertook some experiments to test the truth of the infection of the soil, the results of which elucidate the toxic nature of the bacillus. He found (1) that a bacillus, in some respects resembling in its appearance that met with in the human body, was found in the soil of Hong-Kong during the plague epidemic; (2) that the bacilli of the soil and body occasionally behaved similarly in culture experiments; and (3) that the culture specimens of soil bacilli were, however, innocuous, possessing no virulence when injected into mice or guinea-pigs. Dr. Yersin further found that in his culture of human plague bacilli there were microbes of different virulence, some mildly powerful and some altogether powerless to infect either mice or guinea-pigs. Dr. Lowson, in conjunction with Dr. Takaki, Dr. Kitasato's assistant, found a bacillus in the soil of the garden near his house which closely resembled the human plague bacillus, possessing most of its microscopic attributes, but showing none of its toxic properties. The district whence the soil was obtained lay within the plague-stricken area, but considerably removed from any infected house.

The conclusions to be deduced from the above rehearsal are :—(1) That plague bacilli vary in size; (2) that cultivation experiments produce bacilli identical in appearance but possessing different toxic and infective powers; and (3) that a somewhat similar bacillus is found in the soil (whether at

all times or not is not known) which resembles the non-virulent form the result of bacteriological culture.

Infection of the soil.—The preceding remarks upon finding the bacillus in the soil are pregnant with suggestion. Whether the soil bacillus be virulent or not, whether it be completely identical with the human bacillus met with in plague or not, the fact of a microbe having on the one hand a close resemblance microscopically, and on the other hand a structure and behaviour analogous with certain of the non-virulent culture products of human plague bacilli, is sufficiently important, and brings the elucidation of the matter within the sphere of practical investigation. The dominant idea, before even the bacteriologist or microscopist studied the matter, was that the infection arose from the soil, and there seem no facts to go upon to reject the creed. The Chinese grade the scale of animal infection by their height from the ground, and the modern scientist lends some weight to the suggestion. The medium of the soil may be to the infection of animals as the medium of water bearing the carcase of the dead mosquito is to the filarial-infected human being. Not only may it serve as a mere vehicle, but as a host, in which the parasite passes through a stage of its evolution. Bearing closely upon this subject is the question of the *medium* of spread of plague. I previously discussed this shortly when dealing with contagion. Contagion—that is, intimate prolonged contact, as mother or nurse and child—no doubt is a factor, a potent factor, it may be the only factor, in the transmission of the disease, but few admit it to be more than a small element in distributing plague broadcast over a community. The slow progress of plague and its peculiar paths of selection must be considered if the subject is to be fairly dealt with. Plague is perhaps the slowest travelling disease known; it may take months and even years to extend a few miles. From the City of London it took nine months to reach Soho, and more recently it took ten months to travel from Hong-Kong to Macao—a distance of thirty miles. The lines of communication will not explain this, for in Macao thousands of persons sought shelter in 1894, when Canton and Hong-Kong were plague-stricken. Steamers and Chinese junks and fishing-boats passed freely between the ports, yet Macao did not become infected until nine months after Canton and Kong-Kong were pronounced clear. How did the parasite reach Macao—by sea from Hong-Kong or by land from Canton? If by sea, in all probability it would have been in Macao long before. If by land, how did it

travel—by human or animal carriage, or by the soil? If by human beings, it means intimate prolonged contact of the carriers; if by the soil, it means a bacillary infection of the soil, spreading from the centre and extending by a continuous margin of active growth along paths favourable to its nutrition. As a gangrene spreads from a centre, or a patch of ringworm starts to spread and gradually involves an ever-increasing zone, so may the soil become sown with the germ of plague, and animals that have their habitat thereon fall victims. Some show of testimony towards such a view is afforded by the fact that plague confines its working to the Old World, and does not cross the ocean, and travels backwards and forwards in the belt of its selection; but it cannot be the only means of travel; otherwise, how did the islands of Britain and the Canaries ever become infected at one end of the Old World, and the islands of Hong-Kong and Formosa at the other? Were the soil only the producer or infector, all islands in the area ought to be immune. This not being so, it comes that the poison must be carried by human beings, by animals, by clothing, or by food. That the poison conveyed by them may require the soil as a medium to reproduce the infecting bacillus may be quite possible; but that some other medium than the soil is capable of transmitting the disease is quite evident. As noticed above, the bacillus found in the soil, according to Dr. Yersin, agrees in most points with the plague bacillus, except that, when introduced artificially into the bodies of animals, it is non-virulent. This may indicate, of course, that they belong to another group of the bacterial family altogether.

The Accompaniments of Plague.

Climate.—The weather does not escape blame for a share in the presence of plague when it rages in the midst of a community. Be it dry, the drought is mentioned as a cause; be it wet, the floods which ensue are held to have damaged the crops and the grain supply is spoiled, or the miasm resulting from the subsidence of the swollen streams is connected with the outbreak. In south-east China the epidemic of plague developed at the end of a prolonged and exceptionally dry season. The rainfall was so deficient that most of the wells throughout the district were exhausted, and the crops suffered severely. Without driving the argument of drought too deeply home, it must, however, be observed that, in Bombay, we find a

similar condition of things before the present outbreak. The rainfall over a wider area of country has been so greatly deficient that widespread famine has followed. Without recording any more examples, we must consider whether there is any probability of connection between drought and plague. The bacillus of plague flourishes best in a warm, moist atmosphere; dry, hot air kills it. In many marshy countries, however, the epidemic has been found to disappear the moment the rain ceased and the dry weather set in. In Mesopotamia and in Egypt the epidemic dies out in summer, when the nights and days are more alike in temperature, and the fall of dew is therefore less pronounced. The absence of rain means, of course, dirt in the houses, in the streets, and on the clothes and bodies of the inhabitants; and dirt is held to be the foster-parent of plague. But the absence of rain means dryness of soil; and if the bacterium is dependent on a sufficiency of moisture, it would seem as though a small quantity satisfied it.

Rain.—In Hong-Kong it was hoped, in 1894, that with the advent of rain the epidemic would abate. The rains delayed their onset until well on in May, by which time the epidemic had got a hold in the town. With the rains came an accession—a marked accession—of the number of plague cases. The flushing of the drains and gutters, and the replenishing of the wells with a plentiful supply of water, were attended by an increase in the outbreak. Before referring this positively to the effect of procuring a more moist nidus for the bacillus to develop in, the effect of rain on the habits of the people has to be noticed. In all warm climates a number of the male portion of the inhabitants sleep out of doors—on the roof, in the street, or in verandahs. The effect of rain is to drive all such sleepers within the house, to shut the windows and doors, and to generate all the evils of overcrowding. The good effects of the flushing of sewers are undone, in the opinion of the climatological argument, by the overcrowding which it causes.

Heat and Cold.—In Canton the plague commenced when the temperature averaged about 60° F., and continued when the average daily temperature reached 90°. Further, it must be noted that the disease *disappeared* when heat and moisture were about 90° throughout the district; so that, with regard to the common statement that it thrives best at a temperature between 80° and 85°, it must not be forgotten that, whilst thriving, it also disappears at

the same temperature. It dies out when the conditions which we believe to be suitable to its propagation, heat and moisture, are at their best. But heat does not seem to be an essential to the appearance of plague. In Russia it has appeared again and again in winter, with deep snow on the ground, and the thermometer in the neighbourhood of zero. In Astrakan, in 1878, the disease raged during severe winter weather. We are at a loss, therefore, to connect warmth with plague, or cold weather with its check. We may experimentally show how the bacillus behaves under the abnormal medium which must necessarily surround it in the microscopic field, but that in its natural state it is so affected we have direct antagonistic proof.

Overcrowding.—The argument of overcrowding might be at once set aside by the sweeping statement that, just as plague flourishes in crowded cities, so, likewise, it flourishes in barren deserts, where the population is sparse in the extreme. An analogous disease, typhus fever, behaves in well-nigh the same way, and typhus fever is looked upon as the direct outcome of overcrowding. Now, overcrowding may take place in a country house as readily as in a town house, and the poorer the country the greater the overcrowding. Typhus fever prevails in isolated houses on the west coast of Galway, where the air around is of the purest; but eight or ten people or more in one room, not only sleeping there for warmth, but residing there all day when the weather is inclement in winter, will foster any disease that foul air is capable of producing. As with typhus fever, so with plague. The rains in warm climates drive the inhabitants within their poor dwellings, the snows of winter do the same in colder climates, and the ailments attending overcrowding are most likely to appear. In other countries, as in Mesopotamia, the cessation of rain brings the cessation of plague, as the people can issue forth to sleep, and open their casements, without fear of being drenched. The close, intimate contact believed to be necessary for the propagation of plague is therefore brought about by two entirely opposite conditions which have overcrowding in common; and, taking all the effects of weather into consideration, it would seem that not the meteorological conditions, but the mechanical results of weather generally, are to be reckoned with. Castaldi's account of a dwelling-house in the highlands of Kurdistan, where one might expect that fresh air would be plentiful and overcrowding unknown, confirms the above statements.

"Whatever is most afflicting in poverty, whatever is most revolting in filthiness, is accumulated, as if designedly, around these dens in the interior of which live—or, rather, vegetate—from fifty to sixty men, women, and children." Perhaps the effects of overcrowding are more frequent in the colder latitudes of Western Asia and Eastern Europe, than anywhere else in the world.

Poverty.—Plague is ever associated with scarcity and want. Sometimes it is famine caused by the destruction of crops by floods; sometimes by drought; but from whatever cause plague and scarcity of food are frequently allied. Not only is the grain scarce, but it has often been commented upon that it is bad, musty, covered with blue mould, and fermented. The two conditions may be to each other as cause and effect. The grain is gathered before it is ripe owing to the people's necessity; and the unripe grain not only yields insufficient nourishment, but is apt to undergo fermentation, and be attacked by vegetable parasites. In South-East China there was no question of famine before the 1894 outbreak, but the price of grain was high. This was caused by the prolonged drought in the district, and by the failure of the crops in Siam—Bangkok, the capital of Siam, being the chief source of rice supply to Canton and district. The rice was subsequently obtained at a fairly cheap rate from Saigon, the capital of Indo-Chine, and the price of grain fell. In India at the present moment a serious scarcity prevails, and famine threatens. The prolonged drought is the cause of the failure of crops, and, moreover, the consumption of diseased grain is universal. Three elements are in evidence—drought, famine, and diseased grain; but they are really co-partners and inter-dependent. No one can be held up as the cause to the out-voting of the others. It may be none of these have aught to do with the presence of plague; as it may be argued, plague exists when famine is unheard of; but the combination, which also means overcrowding of houses, must be a factor, if not the chief factor, in the propagation of the disease. In the towns, and in the poorest parts of the towns, would be the most likely spot where the combined effects of want and overcrowding would show; and it is invariably in such neighbourhoods plague develops and stays. In the densely-packed cities of Asia the poor exist for ever on the fringe of destitution, and the least rise in the price of food brings scarcity; so that the term "the poor's plague" holds good for all time.

Sanitary Conditions.—The preceding remarks prepare the way for the recognition of all possible sanitary evils and neglects as accompaniments of plague. Chinese towns are at all times ready to generate any filth disease, and Hong-Kong itself is but little better. The city was hastily built about fifty years ago, when, all too hurriedly, tens of thousands of Chinese sought occupation and habitation under the British rule. From that rush of people, and feverish haste to build without proper control, the city never recovered, and to this day presents sanitary aspects of the most flagrant description. The public sewers are well built on the separate system; the water-supply is most excellent in quality—although, when rains are delayed, deficient in amount; the public latrines are under Government control; the removal of excreta is by the bucket system, and is admirably controlled. Here, however, the bestowal of praise must cease, for beyond them there is much to condemn. House overcrowding is rife, and surface crowding to the extent of 2500 persons to the acre holds sway over the chief area of the native quarter of the town. Dr. Lowson states graphically what is known to every resident in Hong-Kong: "The majority of the houses of the infected district being in a most filthy condition . . . when to a mixture of dust, old rags, ashes, broken crockery, moist surface soil, etc., is added fæcal matter and the decomposing urine of animals and human beings, a terribly insanitary condition of affairs prevails." At another part of his report Dr. Lowson remarks: "To over-paint the pitiable surroundings associated with plague work at the commencement of the epidemic would be impossible. I have entered a long, low cellar, without any window opening, and with the air entering only by a square open shaft from the level of the roof three or four storeys high. Down one side of the shaft ran a broken earthenware drain-pipe leaking freely, the contents streaming down the wall of the air shaft to a shallow pool of filth which crossed the undrained floor of earth. Although it was broad daylight outside, a lantern was necessary to see one's way. On a miserable sodden matting soaked with abominations, there were four forms stretched out. One was dead, the tongue black and protruding. The next had the muscular twitchings and semi-comatose condition heralding dissolution. In searching for a bubo we found a huge mass of glands, extending from Poupart's ligament to the knee-joint. This patient was beyond the stage of wild delirium. Sordes covered the teeth, and were visible

between the parted and blackened lips. Another sufferer, a female child about ten years old, lay in the accumulated filth of apparently two or three days, unable to speak owing to the presence of enlarged cervical glands. The fourth was wildly delirious (the conjunctivæ intensely congested), and was constantly vomiting. The attendant (*sic*), the grandmother of the child, had a temperature of 103° F., and could only crawl from one end of the cellar to the other. She was wet through, and was herself doomed. This is no fancy sketch, but a true picture of how we found some of our patients at the outbreak of the scourge in Hong-Kong." The evidence from Bombay is similar to the Hong-Kong report, and but confirms the state of overcrowding and filth to which Oriental cities, even when partially under British control, may attain.

Caterpillars.—At the time the plague broke out in Hong-Kong and Canton, the district was infested by a caterpillar which covered the trees in myriads, and destroyed the foliage of many. So threatening was the pest, that the Colonial Government in Hong-Kong granted a reward of so much per basket to the collectors of these animals. The accident of their presence may be fortuitous merely, and have nothing to do with the presence of an infective bacillus; but there can be no doubt that the destruction of plant life on the mainland of China from this cause had something to do with the high price of grain which prevailed at the time the plague broke out.

Diseased Grain.—That one is justified in looking to grain as a source of infection, we have collateral confirmation from other diseases arising from grain. 1. Ergot of rye—the mycelium of Claviceps purpurea infecting common rye—is capable of producing epidemics attended by a great mortality. In France, Russia, and Sweden, even during the present century, outbreaks of what the Germans style "Kriebelkrankheit" have occurred. 2. The pellagra of Lombardy is a disease of modern times, but corresponds closely in time with the introduction of an inferior kind of maize as the staple food of the peasantry. This disease is at times epidemic, and although it can be traced to want of care in the drying and preparation of the grain, once it breaks out in a neighbourhood it takes hold of members of the community who feed on the best kind of materials. 3. Acrodynia has so close a resemblance to ergotism and pellagra that it is presumed to be connected with bad grain, although the connection has not been traced. 4. Pelade—a disease met with in Columbia, South

America—is traced to the ergot parasite of maize. 5. Actinomycosis is due to a fungus—probably of the order Cladothriceæ—is even better known than either of the preceding, and the general evidence is that it is introduced with the food. 6. In the uplands of Kurdistan, the four following conditions co-existed or followed in sequence during the 1870-71 outbreak: (1) bad grain; (2) ergotism; (3) epizootic amongst sheep: and (4) plague. 7. At the present moment in Bombay diseased grain is plentiful, and it would seem that Dr. Watson has succeeded in proving a connection between plague and diseased grain. With the help of modern methods of investigation, and the accumulated knowledge of the disease gathered during the last few years, the matter should not be difficult to settle. These instances might be multiplied, but they are sufficient to show that the search for the cause of plague in grain is not a blind hunt, but has creditable precedents.

Differential diagnosis.—In all cases nowadays the microscope is regarded as the one test whereby to test the presence or absence of plague. Rightly or wrongly, this conclusion has held good ever since Kitasato discovered the bacillus in June 1894. The names given in an earlier part of the paper indicate the diseases held to be closely analogous to plague. They are "typhus", "adynamic typhoid", and "intermittent fever"; but the word "bubo" is affixed to all. Still, the name gives the drift of its application, and enables us to apportion its place in the category of disease.

Typhus fever.—The resemblance to typhus fever in many signs, symptoms, methods of spread, and circumstances of development, is worthy of more than a passing remark. It has been noted how overcrowding, whether in town or country, is a potent factor in plague as in typhus fever. Hæmorrhages—so characteristic of typhus fever—are so frequent in some outbreaks of plague as to occasion the use of the synonym of "hæmorrhagic fever". The infectious nature of the two diseases appear so similar, so markedly "catching" to all inmates of an infected house, so sparing in their seizure of visitors. Such instances of the resemblance between the diseases might be multiplied, with the results that we might become converts to the opinion expressed by Murchison, that "Plague is perhaps the typhus of warm climates; the two diseases being generated from similar causes, and differing only in intensity from the effects of climate and other collateral circumstances." The specially characteristic sign of plague is the presence of buboes; the specially determining sign of

typhus fever is the rash. Both may be absent in either; or we may find buboes in typhus fever and petechial eruptions in plague. The eruptions met with in the Hong-Kong epidemic were never characteristic, although many cases showed spots on the skin. These, in most instances, were no doubt mosquito bites, but the occurrence of a pink blush around them gave a specific appearance to these petechiæ. Further, the distribution of typhus fever corresponds in many ways with that of plague. Typhus fever has a southern limit, not being met with within the Tropic of Cancer in either the old or the new world. Typhus fever has never reached Australia or New Zealand, or appeared south of the Equator. Typhus fever, however, is accredited to have reached the United States and Canada, where plague has never been. With this solitary exception, the distribution of the two is synonymous. The pursuance of this subject would lead me far beyond the limits of time at my disposal; but there can be no doubt that we have two diseases generated by a common cause, overcrowding and personal filth; and, if we assume the supervention of a specific microbe upon such states, typhus fever or plague, as the case may be, may arise.

I have yet another disease to add to the list which may be mistaken for plague. On June 26th, 1894, when the plague was at its height, I saw a Parsee patient dwelling in a house in which plague existed, suffering from fever (104° F.), dry tongue, headache, backache, and large swollen glands in the left groin which had suddenly appeared. Plague seemed the only diagnosis, and the man, much against his will, was sent to the plague hospital. In two days he came back again quite well, and on examining him I found his urine thick and milky. That night I found filariæ in the man's blood, and knew I had made a mistake in the first instance. Of course, the mistake is most likely to happen, but nevertheless it is not pleasant to think that we had subjected the man to the terrible danger of plague infection.

The conclusions drawn from a study of the spread of plague are:—I. Varieties: 1. The varieties of plague known under the names (*a*) fulminant, (*b*) typical, and (*c*) pestis minor, are allied. 2. The cause of fulminant and typical plague is a diplobacterium in the blood and tissues. The cause of pestis minor may be an allied diplobacterium, but with a lesser toxic power. 3. An appropriate name for the fulminant and typical plague is "malignant polyadenitis." An appropriate name for the mild variety (pestis

minor) is "benign polyadenitis". II. Infection and contagion: 1. Plague is infectious chiefly by the dust arising during the cleansing of dwelling-houses which plague patients have occupied. 2. Plague is contagious by prolonged and intimate contact with the plague-stricken, as in the case of a nurse carrying a child ill of the disease. III. Distribution: 1. Plague is met with in a definite area of Asia, which may be termed the "plague belt". 2. That the home of plague at the present day is Mesopotamia and the countries adjacent. 3. From Mesopotamia as a focus the plague may spread northwards to the Caspian Sea, westwards to the Red Sea, southwards as far as Bombay, and eastwards as far as (Formosa) the China Sea. 4. During the present century plague has shown a western retrocession and an eastern accession of virulence. IV. The bacillus: 1. Typical plague (malignant polyadenitis) is associated with pestis minor (benign polyadenitis). 2. A bacillus of somewhat similar appearance microscopically is reputed to be found in both.* 3. The bacilli differ in their toxic powers only (?). 4. A benign polyadenitis may run its course without being preceded or followed by the malignant variety. 5. Malignant polyadenitis may run its course without being preceded or followed by the benign variety. 6. The bacillus of the benign variety attains malignancy by passing through some intermediate host, possibly, but not probably, the rat.

At the conclusion of Dr. Cantlie's paper the following remarks were made by Dr. Lowson, of Hong-Kong, who, at the request of the Council, has consented to their publication in the *Transactions*:—

"The origin of the plague bacillus cannot be discussed here, any more than can the origin of man. That the virus can originate in the soil, as some people persist in believing, I do not think possible. It is useless now to add evidence of the predisposing causes of plague; suffice it to say that overcrowding and insanitary filthy surroundings generally were well marked in Hong-Kong, Macao, and Canton, where my knowledge of the disease has been acquired. I wish to discuss shortly the question of infection of the soil. In Hong-Kong, in Taipingshan, there were numerous earthen-floored houses which were shut up owing to plague cases having occurred in them. These houses, however, were not so numerous as has been generally stated,

* This is under discussion now (December 18th, 1896).

the large majority of tenements being floored with unglazed tiles or bricks. Yersin took earth from one of these earthen-floored houses under my supervision, at 7 in., 18 in., and 24 in. depth. He said he found the bacillus in earth taken at a depth of 7 in., but afterwards qualified his statement, through Dr. Cantlie, that he had found it not deeper than 3 ins. Matters thus being in a somewhat doubtful state, I asked one of Dr. Kitasato's assistants, Dr. Takaki, to conduct a series of experiments with me on the subject. We could not find the plague bacillus in the earth by culture; we could not kill rabbits or rats of plague by the culture got from the earth; we could not kill them by plague by the earth itself direct; none of them suffered from plague to all appearance. A bacillus was found in the earth somewhat like the plague bacillus, but smaller and distinctly dumb-bell shaped. This same bacillus was found in our garden, where plague germs were not likely to have been introduced. This evidence leads one to conclude that at least in the houses we examined there was, *in all probability*, no plague infection of the soil. Before Dr. Takaki went to Japan, numerous other specimens of earth were taken at depths of from 1 to 6 ins. from the aforesaid earthen-floored houses, these specimens being examined, I understand, in Kitasato's laboratory, and I was informed that they had given no positive results as to plague. I do not of course affirm that plague infection of soil cannot take place; but it seems to me that in Hong-Kong such infection was improbable. I would add, however, that in a Chinese village there is a possibility that the superficial layers of the soil will in time get infected. The question requires further proof, and from an independent worker. Yersin believes in an infection of the soil, I can only at present believe in a superficial infection of the floor in an epidemic. The deeper infection I cannot believe in. If the latter is proved, then the question of regeneration of the bacilli has to be proved, before one can fairly blame the earth for being the root of all evil in plague.

"On the question of introduction into a new locality, the carriage by fomites should be paid more attention to than has previously been the case. That it can be thus carried by clothes is, I think, undoubted, and I would add two cases in support of this view. In 1894 the Chinese 'boy' of one of the volunteer search party was admitted to hospital with plague. This 'boy' stayed at the Peak, far outside the infected areas, and the *only way we could account* for his attack was, that he was in the habit of cleaning the clothes

which his master regularly wore whilst at work in the infected areas. In 1896 the washerman at Kennedy Town Hospital died of plague, and the only chance of his having been infected by other means than by the bedding and clothing, was his possible exposure to infection for a few hours, *eleven* days before the commencement of the attack, and even then it could *not be proved* that he was ever near any infected place. Eleven days is also at least two days beyond *the longest period of incubation known, which is nine.* The carriage of infection by human beings is not so well marked over long distances, sea air and the more sanitary conditions of ships being against its development, even after a case has occurred on shipboard. It must be remembered also that the people who would carry infection belong, as a rule, to the poorest class, who cannot afford the luxury of running away on a long sea voyage. In 1894 no one was discovered suffering from plague at Shanghai on steamers arriving there from Hong-Kong, and only one case was found at Singapore. In 1896 I do not know about this matter, but up till May, very few, if any, were found at Shanghai, only one in Japan, and three in Borneo. However, on shorter journeys, which can easily be taken by those poorer people, the villages around Canton and Hong-Kong became much more rapidly infected. As far as I can ascertain, Canton was well infected in the beginning of February 1894; Hong-Kong in the beginning of May 1894; Macao in February 1895; Swatow and Amoy in March 1895; whilst Formosa has become the seat of the disease in 1896—probably about March. To us in Hong-Kong a question of great interest was the condition of affairs in Singapore. Last winter, 1895-6, the death-rate of Singapore went up to between 60 and 75 per 1,000 per annum for several weeks, 'fever' being the presumed cause. Two cases of plague came to us from different ships which had just arrived from Singapore. One was a Chinaman who had left Hong-Kong and Amoy for Borneo, Bangkok, etc. The vessel's crew, some five weeks before, had called in at Singapore, and on the voyage to Hong-Kong the man in question had been seized by symptoms which were undoubtedly those of plague. The other case came from Singapore on a ship, and was landed in Hong-Kong. Another attack occurred on one of the Calcutta boats. The ship arrived at Singapore eight days after leaving Calcutta. Four days after leaving Singapore this man, a steward, who had been ashore in the slums of Singapore first became ill, and landed in Hong-Kong with typical

plague symptoms. This case seemed to me conclusive that Singapore had some plague germs about. An extremely long incubation period might be possible, and also an infection from Hong-Kong of almost two months' duration; but with my experience of incubation periods in plague it is hard to believe that Singapore has not had some plague cases. And where did the second Chinaman pick it up? The authorities at Singapore, however, have hitherto been unable to discover the presence of the disease. Against ship infection it must be remembered that all ships in the far East, since 1894, have been most carefully looked after by their owners in the matter of cleanliness and disinfection, and this fact alone makes it improbable that the above patients (one Chinaman and one Indian) took the disease with them from China in the first instance. Chinese habits in regard to clothing are peculiar, and large quantities of plague-infected clothing must now be going about the country there, as it is not usually destroyed. The clothes of all patients in Hong-Kong during the epidemic in 1894 were destroyed, and in 1896, partly destroyed and partly disinfected by steam. In 1894 I counselled the disinfection of all clothes in pawnbrokers' shops in Hong-Kong (which are simply magnificent breeding-grounds for disease) for protection against small-pox and other diseases besides plague, but I regret to say that nothing has been done. In China it is the custom to put one's winter clothes in pawn at the beginning of summer, and resume wearing them in the winter; and, although without definite proof, I believe that this had something to do with the disseminated outbreak in Hong-Kong in Dec. 1895. The first cold spell came about Dec. 18th, and it was remarkable that within a fortnight we had fairly commenced on our 1896 epidemic, in which there were about 1,200 cases. In Canton there was the superfluous clothing of most of the 100,000 or thereabouts that died in 1894: what damage this has done in the surrounding country I must leave you to guess. I am sure that if such a thing happened in England—I mean the non-disinfection of clothes presumably partly infected—the matter would not be allowed to rest where it is simply because of its magnitude.

"I do not believe the poison can be carried by the atmosphere unless in its most condensed form, and over only short distances. Fresh air and sunlight are the greatest enemies of the bacillus. If the disease were easily carried by the air, large tracts would be affected, whereas its

habits already have proved that it proceeds along the different trade routes.

"A few facts in connection with the question of infection of animals may not be out of place. First as regards rats: statements have appeared regarding the Hong-Kong epidemic which should be contradicted. My duties and residence necessitated moving about in what were to be the infected districts in Hong-Kong more than most people, and I never saw those dead rats which were supposed to be present. Moreover, the same statement was made about Canton and Macao, and from enquiries which I made there, it all resolved itself into the same thing—no one could give any reliable evidence; in fact it was all negative. In these places the infection of human beings and rats was, as far as I could ascertain, simultaneous; and it should be remembered that I looked at the matter from an impartial standpoint, and more as a commissioner in those cities than as a witness, as I was in Hong-Kong. One must also bear in mind that the incubation period is short in those animals, and where man usually dies in from 4 to 12 days after infection, rats usually do so in from 36, 72, or 96 hours. A natural consequence will be that, after an epidemic has begun, the death of rats will be more noticeable than the death of human beings, not only on account of the shortness of the incubation period, but also because rats generally come out of their holes to die, whilst human beings usually die out of sight. Under this heading your attention should be drawn to a most important point:—Disinfection of houses can never be complete where rat-holes are left undisinfected, and where floors cannot be torn up to disinfect underneath. Many of these subterranean passages and haunts must remain infected for some time, and when new generations of rats arrive on the scene, it is more than possible that fresh epidemics will spring up. With regard to pigs, the following experiments may be interesting. In 1895, four young pigs were fed on plague spleens for some time. None of these died, or were apparently affected. Later on they were inoculated by culture and implantation of plague bubo, when a slight elevation of temperature was observed for three days, but they returned to complete health. In 1896, however, one pig experimented on by Dr. Wilm, at Kennedy Town Hospital, sickened and died twenty-two days after last partaking of plague spleen. The temperature during this time was only slightly above normal, and the animal took food fairly well, and no particular notice was then taken of it. To our surprise, it

was found dead on the twenty-third day, and a post-mortem and bacteriological examination proved that it had died from what was evidently subacute plague. The bacillus was found in the enlarged mesenteric glands, whilst the other glands presented appearances of enlargement and inflammation, but not so markedly hæmorrhagic as affected human glands generally are. It may be added that shortly after this, a ship arrived at Hong-Kong from Pakhoi with a cargo of pigs. Disease broke out; the animals affected were segregated. Some died, the others were killed. They were found to present the same appearances as the one mentioned above, and an *almost similar bacillus was present.* Dr. Wilm investigated this, as I was at other work; and as it happened just before leaving on holiday, I cannot give a complete report on it. However, if the similarity of the disease be established, we enter a new domain of argument; infection by food. Several observers of the pig disease contended it was *swine erysipelas;* so that at present it is better to wait for more definite information. Cattle were treated in the same way as pigs, with the result that after implantation of bubo, the temperature rose on the second day to about 107°, and gradually fell within forty-eight hours to normal, the calves showing no other general symptoms. The place of inoculation was surrounded by considerable œdema, and when the serum from it was examined on the fourth day no bacilli could be found in it. Two calves were experimented on frequently. After each inoculation the temperature went up as before, but not to the same height. Ultimately the temperature never rose after this implantation, and I proceeded to try some experiments with their blood serum. It was tried twice on Chinese without the slightest effect—the disease being in too advanced a stage. In two cases where experiment was made on rabbits the disease seemed somewhat less severe, as death did not occur until the eighth and ninth days—death usually occurring on the third or fourth day —and the temperature chart showed a lower average height. Guinea-pigs and rabbits always die rapidly after infection. Dogs and cats I have not yet experimented on, but they seemed to escape entirely in the epidemic, no dead dogs or cats having been found by the search parties. Pigeons and fowls escaped altogether, and I have been unable to transmit the disease to them. Kitasato also experimented on fowls, with a similar result. Grain has been frequently blamed for conveying the disease in India, and experiments have been undertaken in this

direction; but it seems to me that at present the idea of grain infection is somewhat far-fetched, and very many objections will have to be overcome before grain can be proved to be a vehicle of plague. Infection by the alimentary canal has been found possible, and in future a great amount of attention will have to be paid to this and to the food and water supply. It may be interesting to point out several variations in type which I have noticed in connection with the disease in Hong-Kong. One thing there has practically been no variation in: that is, the Chinese death-rate, which has been 93.95 per cent. of Chinese affected. The death-rate of those admitted alive to hospital being 80 per cent., practically the same all along. In 1894 there seemed to be many more cases of inoculation than in 1896. Dr. Wilm, who has been assisting the Government medical officers, has come to the conclusion that the disease is chiefly intestinal in origin; but this has only been a feature of the 1896 disease, where the mesenteric glands especially seemed to be infected. Noticeable glands were far more numerous in 1894 than in 1896. In 1894 large quantities of albumen in the urine were never met with in my experience; in 1896 it was a common occurrence, and casts were often detected—in fact, nephritis was a marked condition in 1896. In 1894 convulsions were not infrequent, and were far more common than in 1896, when they were seldom, if ever, met with. These convulsions in the first outbreak were sometimes extraordinary in violence. Intermission of pulse was very common in 1894—not so in 1896. Sudden heart failure was also much more often met with in 1894—by this I mean a man might have a full and bounding pulse, with apparently a good chance of living a few days at least, and yet be dead of heart failure in five minutes. In the latter year (1896) the heart failure was much more gradual. The mesenteric glands, although always slightly affected in 1894, were, *as a rule*, more affected in 1896. In fact, the difference between the epidemics seemed to be that, in 1894, the head and heart symptoms were prominent, in contrast to the mesenteric and nephritic symptoms present in 1896. The dusky plague countenance was much more noticed in 1894 than in 1896. While on this part of the subject, I would call attention to the 1665 outbreak in London. The *tokens* generally spoken of by Boghurst, Defoe, and other writers, are undoubtedly petechial; and, after consideration, I have come to the conclusion that at that time the disease was evidently petechial in type, the condition of the skin

and smaller blood-vessels evidently supplying one of the most prominent symptoms of the disease, whilst hæmorrhage from the bowel and urinary tract was concurrent. These latter hæmorrhages have been remarkable because of their almost entire absence in Hong-Kong. Convulsions are spoken of sparingly indeed by Boghurst and Defoe. In Vetliauka, in 1879, apparently a lung type of the disease prevailed, the same as in 1346 in Europe.

"Several old authors have spoken on the tendency of 'all diseases to become plague' during epidemics of plague. Some of these have been proved to be wrong by writings of their contemporaries, and certainly in Hong-Kong there was no such tendency. The Government Civil Hospital was just as full as usual with ordinary diseases, and the deaths from these diseases showed no change; of course, where plague bodies predominated so much there was a tendency at the time to overlook the fact that deaths from 'natural causes' were going on as usual. One curious fact remains, however, that the death-rate for the year was only very little above the average, the latter part of the year showing a great diminution: probably due to the 'weeding-out' process of the disease, and the flight of many diseased persons to the mainland, who were in fear of falling into the hands of the 'foreign doctor devils'.

"A few weeks ago telegrams from Merv stated that 10,000 inhabitants had been killed by some malignant fever—I wonder if it was plague? Cholera and small-pox would be soon recognised, as also would typhus, and it may, therefore, be not unlikely that plague is the probable enemy. Whilst speaking of infection of ports it may be interesting to know that, in connection with the Bombay epidemic, Hong-Kong should be looked on as a probable source of the disease. In 1894 and 1896 several Indian shops in Hong-Kong were closed on account of plague; and two shops in particular, I remember, were closed, which did a large amount of business with India. If Merv has been suffering from plague, I think it very improbable that Bombay has been infected from there; if it had been we should expect to have had a train of it through Afghanistan.

"A few words on the question of ambulatory plague: I have seen practically nothing of it. In the Government Civil Hospital records of the months preceding the epidemic, I could not find one case which could by any possibility be put down to this cause. During the plague period I never saw a *bonâ fide* case. Everyone discovered glands in their groins at that time; and policemen and others

who were suffering from enlarged inguinal glands, of course tried to make out that it was plague they suffered from, and not a much more common malady. In 1895 and 1896 I do not think I saw a single case which could possibly be put down as ambulatory plague: I heard of several—I wish I had seen them. In Swatow, Dr. Layng stated that several cases had come under his observation. I do not doubt for a minute these cases occur, but it is a subject on which observers are inclined to make errors, and glands only become entitled to special attention when rumours of plague are current. Epidemic virulence seems to vary very little amongst Chinese, so far as the mortality goes. From our experience in Europeans it is evident that Western races are not so easily knocked down by it, and one can understand how Boghurst got 75 per cent. of recoveries in 1665. In 1894 our European death-rate was 18 per cent. In 1896—I have not the figures by me—I think it was slightly higher. Indians, Manilamen, and Eurasians have nearly the same mortality as Chinese—in 1894 really higher, as all of them died.

"We now know that the poison is given off by the discharge from buboes, by the fæces, by the sputum, and frequently by the urine. Cultures from the bubo, fæces, and sputum almost invariably kill rabbits and guinea-pigs. In cases of well-marked albuminuria, cultures from the urine lead to the same result. I do not think that the poison is given off in ordinary respiration—the disease being more like phthisis in this respect—the sputum contains the bacilli but not the breath. The poison lies mostly in the floors of houses, multiplying in the dirt; and it is well to note that of all our search party, and people whose actual work was not concerned with the cleaning out of the houses, not one got plague, and they certainly respired enough of the air in infected houses in their bi-daily two-hour search. The "whitewash brigade" and the cleaners were those who suffered—those who had to stir up the dirt and dust on floors and furniture. In the Tung Wah Hospital only one attendant died from plague, and that was in the slaughter-house branch, where hygienic arrangements were perfect. When they were in the glass works the atmosphere was extremely foul—horrible beyond description—and yet none of those who stayed there as attendants contracted plague, and they used to be in attendance on the sick for often twelve hours or more at a stretch. The sanitary condition of the floor was carefully looked after, chloride of lime and carbolic acid being sprinkled

freely about before each sweeping out and cleaning. Yet for twenty-four hours at a stretch the Chinese attendants would be in that atmosphere, and nothing happened to them; their sleeping quarters on several occasions being part of the same rooms where the patients lay. I am forced to the conclusion that the mere air round a patient is not the infecting material; it is the dust and dirt which is stirred up and consequently respired which brings about an attack of the disease. In the disinfecting and cleaning squad of the Shropshire Regiment none were affected after the free use of chlorine in the houses was begun, and the fluid left after the action of the sulphuric acid on chloride of lime diluted and freely used on the floor. Again, several coloured men in charge of the dust carts, and who threw the dirt on the fires to be burnt, died from the disease; a fact tending to implicate the dust.

"Another point to note. Some of you may not be aware of the construction of Chinese houses. They are practically solid boxes divided into a ground floor, first floor, and second floor—occasionally a basement. The stairs lead up inside the house, and the partitions, as a rule, permit the freest passage of air. Now, in 1894, it was extremely common for the inhabitants of one floor to be almost all affected, whilst the inhabitants of the other escaped; and this was all the more marked where 'strained relations' between the inhabitants of each floor prevented friendly visits. I understand the experience of 1896 has been almost similar, but of this I cannot give a definite opinion, as I have had no town work since a medical officer of health was appointed in the end of 1895. Surely this is against a purely aërial infection such as is claimed by some writers. None of the attendants who handled the dead in the mortuaries took the disease, neither did any of the coolies who removed the bodies from the hospital wards. The bodies were usually placed in coffins by the feet and pigtails, the 'dead' coolies, as they were called, taking care not to handle the bodies more than could be helped. All this simply points out that the infected houses are the great danger spots, and that it is plague-infected dust and dirt which is to be feared by those working amongst the disease.

"In 1894 the plague-infected areas were localised. Although many cases were found outside these areas, yet the majority occurred in them, and whole floors of people were swept off—sometimes the top floor, sometimes the ground floor, sometimes the middle floor. These were

attacked with conspicuous impartiality, thus showing that the question of proximity to the earth had not much influence in the matter. In 1896 the outbreak was disseminated, and one or two cases in each house was the rule, and I should say the majority were found in the upper storeys. Had the disease been easily propagated by the breath surely this would not have happened: one would have expected the whole household to be swept off. True, some houses were treated in this manner, but a large majority were otherwise. Was intestinal infection the cause of this? The inhabitants of the small Chinese villages in Hong-Kong kept up a rigid quarantine on their own account, driving off all Chinese strangers who approached, and escaped scot-free in 1894; but in 1896, where this was not kept up, several of the villages suffered. The immunity of the boat population was probably due to their comparatively cleanly habits, and living in the fresh air on food which had less chance of being contaminated by the poison.

"The views I have given in Hong-Kong on the subject of infection by inoculation have been discounted by some, because only two instances of lymphangitis were given in my first Plague report. Every case could not be described, but the number of cases of lymphangitis were numerous. Besides, it is not at all necessary to have lymphangitis, as the amount of reaction at the site of inoculation and subsequent lymphatic trouble depends on the purity of the infecting material and the susceptibility of the animal. Large numbers died before lymphangitis had a chance to show itself; and I have seen rabbits die from inoculation without the slightest sign of wound reaction, more especially when the site of inoculation was on the foot or leg. Dr. Molyneux, of Ningpo, can corroborate me in this, as it was his custom to go round the wards with the iodine bottle in his hand to treat the lymphangitis. He it was who laid the greatest stress on this lymphatic trouble; but at the time we used to laugh at the pertinacity with which he preached about it, not thinking that future observers would doubt what we told them on this subject. Other diseases are inoculable without lymphangitis; why not plague? In 1896 lymphangitis was rare, but still cases appeared occasionally; and it was my firm conviction that in this latter year intestinal infection was the commonest source of the disease.

"Dr. Cantlie's limits of plague are not, I think, of much value, as plague will go wherever it is carried, and thrive

there if suitable pabulum be supplied. Uganda, in Central Africa, is at the Equator, and plague has been found there, and is there now, although it has escaped all previous authorities. The question as to whether Bombay pigeons have died from plague has not been settled yet, as also whether grain is at fault or not. As regards Dr. Cantlie's remarks concerning the Sister who got plague, too much reliance should not, I think, be placed on them, as there were other means by which the disease may have been contracted. In 1894 many mothers died while suckling their children, who survived and were left upon our hands in hospital to be disposed of. Before finishing, I would commend the use of chlorine to those who have to work amidst plague. It is impossible to cleanse, disinfect, and whitewash every house in an epidemic; in fact we generally march after it, and the organisation of large squads for disinfecting by chlorine, *both before and after houses have been infected, should be the first method of attack* when an epidemic has got a fair start: the cleansing takes up so much time that the disease will simply run over the country before being caught up. This, with written instructions for cleanliness and attention to food and water, should be the first matter to be attended to. The diluted chlorine residue in the disinfecting jars should be used on the floors; it is in this point that chlorine is superior to sulphur in plague. The terrible ravages that plague has made lately in British colonies has been, in my opinion, due to the fact that we have not realised the extraordinary rapidity with which plague can march; or the futility of trying to catch it up when lengthy and laborious disinfection and cleansing has to be carried out with an inadequate staff.

AGE-INCIDENCE IN RELATION WITH CYCLES OF DISEASE-PREVALENCE.

By W. H. HAMER, M.D., Assistant Medical Officer of Health of the London County Council.

(*Read: January* 15*th*, 1897.)

The way, to be presently more particularly referred to, in which the question of age-incidence comes to be of interest in connection with cycles of disease-prevalence may, perhaps, best be indicated by briefly referring at the outset to certain explanations which have been given of the causation of epidemic waves. It is not, of course, proposed to deal with these explanations in a comprehensive manner, or indeed to dwell at all upon any explanation which has no apparent connection with the question of age-incidence. Again, inasmuch as the inquiry concerning age-incidence, the results of which it is proposed to give, has only extended to four diseases, viz., small-pox, scarlet fever, measles, and whooping-cough, and has been instituted with a view to ascertaining whether annual variations in age-incidence accompany the annual variations in the mortality from those diseases in London, it will be well to confine consideration to points which can be illustrated under these limitations.

Two or three references to well-known authorities will serve to show the nature of the questions to be alluded to.

Dr. Arthur Ransome, in a paper on "Epidemic Cycles", read before the Literary and Philosophical Society of Manchester and Salford in 1880, discussed the reasons which determine the recurrence in cycles of years of certain epidemic diseases. Dr. Ransome illustrates the paper with the now well-known charts which he constructed from figures supplied to him by Dr. Berg, of Stockholm, and which so strikingly display the curious behaviour of whooping-cough, small-pox, scarlet fever, and measles, in Sweden, from the year 1774 onwards. Dr. Ransome enumerated seven possible reasons for the remarkable regularity of epidemic cycles observed by these diseases. Of these

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theories three may be particularly referred to in this place.

On one hypothesis, explanation of the recurrence of epidemics after certain periods was to be sought in the fact that "certain years of life are prone to the disease, and that when these have been cleared away the disease could not prevail extensively, until there were again persons of fit age to receive the poison." This simple "age" theory was not, Dr. Ransome showed, capable of explaining the phenomena observed. Another hypothesis was to the effect that variations in "the powers of offence and virulence" manifested by the disease, were brought about in connection with altered phases in the life-history of the disease germ. A third hypothesis, and one which Dr. Ransome regarded as affording a simpler explanation of the facts than any of the others is, "that a certain density of the population at susceptible ages is necessary before a disease can spread with the vigour of an epidemic." Dr. Ransome says, with reference to this hypothesis, "probably all the facts would be accounted for if we suppose that these disorders can only become epidemic when the proximity between susceptible persons becomes sufficiently close for the infection to pass freely from one to the other. Exanthematous diseases rarely attack the same individual twice in his lifetime. When, therefore, an epidemic has, by either a fatal or non-fatal attack, cleared away nearly all the susceptible persons in a population, mostly infants and children up to a certain age, then it must necessarily wait a certain number of years before the requisite nearness of susceptible individuals has been again secured. There must, in the interval, be a gradual re-stocking of the nation with material fit for the epidemic to feed upon, and it can only spread when the requisite proximity is attained, when the meshes of the network of communication are sufficiently close for it to include all susceptible persons in one grand haul."

Dr. Ransome further illustrates in his paper the extent to which this hypothesis is supported by the peculiarities of the different epidemic waves, and more particularly by the contrast exhibited between certain striking features presented by the Swedish curves and the phenomena deduced from the available records with respect to mortality from the diseases under consideration in England.

Dr. Ransome returned to this subject in a paper on "The Form of the Epidemic Wave, and some of its Probable Causes", read on June 7th, 1882, before the Epidemiological

Society. In this paper he distinguished, in the Swedish scarlet fever curve, between the short cycles of four to six years, and the long undulation of fifteen to twenty years or more, which he likened "to a vast wave of disease, upon which the lesser epidemics show like little ripples upon the surface of an ocean swell." He was unable, he said, to give any adequate explanation of the many years' variation of prevalence of epidemics—their "nutation period", as he suggested it might be called. With regard, however, to the smaller waves, he recurred to the explanation which had been already given, and which requires for the spread of an epidemic that—as he now expressed it—the "necessary degree of potency of the poison has been obtained, to enable it to traverse the average distance between person and person."

The question under consideration has, moreover, been referred to by Mr. Netten Radcliffe, in the article on "Periodicity in Disease", which he contributed to Quain's *Dictionary of Medicine.* "In those diseases", Mr. Radcliffe writes, "which are communicable from the sick to the healthy, the influence of an accumulation of susceptible persons will have to be distinguished from extraneous conditions presumably operative in determining the periodicity." The fluctuations of small-pox after the year 1840 in the Metropolis, in their close relationship with the fluctuations in quantity of unvaccinated children, are particularly referred to. Mr. Radcliffe discussed, too, the hypothetical case, in which all epidemics are regarded as being of equal intensity, and no condition is assumed to be operative in determining an epidemic beyond the accumulation of susceptible people. He shows in this case that the cycle of the epidemic will be obtained by dividing the number of persons attacked, or otherwise rendered insusceptible during an epidemic, by the annual excess of births over deaths (all causes) with other increments of susceptible population. After discussing the application of this formula, Mr. Radcliffe says, "It is easy, in these considerations, to find reasons for differences in epidemic cycles among different communities, for differences in the intensity of successive epidemics, and for apparent alterations of susceptibility among communities. And these reasons will deserve to be considered before going in search of other reasons."

In 1892, in a paper on "Measles Epidemics, Major and Minor", Dr. Whitelegge discussed the phenomena presented by measles outbreaks in a number of communities, and

distinguished between the minor epidemics, "occurring every year or two and in a sense mechanically", and the long waves of measles prevalence, which he suggested were due to progressive alterations in the intensity of the measles virus, and which he said might be fittingly termed major epidemics. While holding that Dr. Ransome had rightly laid great stress upon the accumulation of susceptible children, Dr. Whitelegge drew attention to definite variations in the "quality" of measles; and he showed that in certain instances there is observable a parallelism between the rise and fall of epidemic prevalence and the severity of type of the disease as indicated by variations in case mortality. In his Milroy Lectures on "Changes of Type in Epidemic Diseases", Dr. Whitelegge developed in detail these views, and his observations were extended to epidemic diseases as a whole. He divided epidemic diseases into two distinct classes: those which are determined by increased energy of the disease itself, and those which are not; and he showed that whereas certain diseases are of unstable type, and present marked contrasts, both in quality and quantity, "the former dominating, and in a sense causing, the latter," in other diseases stability of type is more apparent, and the analogous changes are slower and less conspicuous, "owing to the fact that other causes besides intensification of the poison bring about epidemic prevalence, and other causes than attenuation arrest it."

Dr. Whitelegge distinguished between "superadded waves" (annual seasonal waves, milk epidemics and water epidemics) and the broader cycles determined by altered quality of the contagion. The superadded waves, he said, were generally attended by a lowered case mortality, because the conditions under which they occur imply a lessened average susceptibility, and therefore a less severe average attack. On the other hand, he pointed out that in all great epidemics, and on a smaller scale in most if not all epidemic diseases, there occurs a change in the quality of the disease itself: "the intensity rising and falling at intervals which are not necessarily uniform for the same disease, and are very different in different diseases." The "intensification", he adds, is marked by greater severity of attack, greater power of overcoming comparative insusceptibility, and greater power of epidemic diffusion.

The mobile diseases are capable of rapid and even abrupt changes in intensity; with regard to other epidemic

diseases, those of comparatively constant type, "there is evidence of a rise and fall of intensity, if the epidemic course be traced for a term of years, perhaps covering several minor epidemics." Again, he says: "In these brief outbursts of diseases of more constant type there is little, if any, change of intensity comparable to that of the mobile class, the prevalence being determined and controlled mainly by external conditions, but the type being that of the prevailing phase of a broad cycle."

Among the illustrations of the evidence of gradual change in diseases of more stable type, Dr. Whitelegge dealt with the mortality curves from scarlatina, small-pox and measles in London. He pointed out that "the scarlet fever case mortality in the London fever hospitals, from 1832 onwards, shows a tolerably steady rise and fall, with wave lengths of five or six years; and, moreover, this rise and fall are at nearly all points parallel with that of the London scarlet fever mortality, especially during the period 1859 to 1880." As regards the statistics of the Metropolitan Asylums Board hospitals which are available from 1872 onwards, he noted that "here again the mortality curve is seen to be moulded primarily upon that of severity, and as long as the severity runs high the mortality curve is closely parallel to it; when, however, the severity declines to a low level, its slight variations are no longer traceable clearly on the death curve, being obscured by the sharp and irregular variations in prevalence, due to season or accident, or accumulation of susceptible persons."

"It seems, then," Dr. Whitelegge said, in summarising the evidence with regard to scarlet fever, "that when the scarlet fever curve rises at more or less regular intervals it does so for two reasons—because the attacks are more numerous, and because they are more fatal in type. They are more numerous because there is increasing power of overcoming resistance whereby those who were safe during the interval become more liable to attack; because the severer cases are more capable of diffusing infection; because the wider diffusion thus brought about lessens the chance of susceptible persons escaping; and lastly, because the susceptible population had to some extent been reinforced during the years of comparative freedom from scarlet fever."

In reference to small-pox Dr. Whitelegge notes, with regard to the London figures, a tendency to slightly greater destructiveness in each successive minor epidemic, culminat-

ing in the great epidemic of 1871. "Whether the minor epidemics which occurred in London at intervals of four or five years were attended with any temporary increase of severity, as in scarlet fever, is a point of considerable doubt. Some indication of such a change is afforded by hospital statistics, though not very clearly." Again, in summing up the case with regard to small-pox he says: "The type of small-pox seems to be stable, rising and falling in intensity if traced through long periods of years, the intensity being indicated even more by the power of epidemic diffusion than by severity of attack; but apart from these long cycles there is a tendency to shorter cycles of four years or so, which may or may not prove to be accompanied by temporary increase of intensity, and are probably governed by the accumulation of susceptible persons and other external conditions."

With regard to measles, Dr. Whitelegge shows that evidence is forthcoming of long waves of prevalence accompanied by increase and decrease of virulence, "each wave lasting often twenty years or more in this country, although perhaps less elsewhere." "As in small-pox," he adds, "the rise and fall of the broad primary curve is interrupted (in a sense mechanically) at short intervals by explosions occurring when the accumulation of susceptible persons is sufficient, and the climatic and other external conditions offer sufficiently small resistance."

It will be seen, therefore, that the doctrine that quality dominates and in a sense causes quantity, of which Dr. Whitelegge gives many illustrations in the case of mobile diseases, is also to be thought of as possibly having application even in the more stable types. The evidence in the case of scarlet fever is of a striking character; it is more doubtful in the case of small-pox; and as regards the shorter cycles of small-pox, Dr. Whitelegge notes that they are probably governed by accumulation of susceptible persons and other external conditions. In measles, too, the interrupting explosions occur "in a sense mechanically". There is, however, even with regard to small-pox and measles, the suggestion that the larger waves of prevalence are associated with alterations of intensity.

The mechanical theory, pure and simple, has thus been shown to need qualification. Hirsch stated it to be his opinion that two factors only "determine the recurrence of an epidemic of small-pox; on the one hand the necessary number of persons susceptible to the morbid poison, and on the other hand the introduction of the virus itself." With

regard to measles, he said, "the same law accordingly applies here as in small-pox epidemics." These statements would now probably be regarded as wanting modification, but it may be noted that Hirsch pointed out that scarlet fever was not to be included in the same category with small-pox and measles; indeed, he specially refers to "variation in the intensity of the disease" as a conspicuous peculiarity of scarlet fever. From any point of view, however, the mechanical theory has still considerable interest in connection with the cycles of the more stable forms of disease.

In the address "On the Study of Epidemiology", which Mr. Shirley Murphy read before this Society at the commencement of the Session 1894-95, he dealt with certain of the conditions which affect epidemic disease, and particularly alluded to the influence of meteorological conditions, to the changes of type of disease discussed by Dr. Whitelegge, and to the question of "school influence". In connection with the last-named subject, he referred to the change in the age-incidence of diphtheria mortality, to which he had directed attention, and which he had examined in detail with the results set out in his Preliminary Memorandum on the Increase of Diphtheria in London. Mr. Shirley Murphy also demonstrated the existence of an "August depression" in the London curves of notifications of scarlet fever and diphtheria, and alluded to the bearing of this fact upon the question of "school-influence". He, moreover, discussed circumstances which influence the fatality of disease, and more particularly referred to variations in the seasonal fatality of scarlet fever and diphtheria. These subjects have been further dealt with in Mr. Shirley Murphy's later reports.

A consideration of the question as to the extent to which the mere mechanical accumulation of susceptible persons has effect in tending to produce the shorter cycles of prevalence, in such diseases as small-pox, scarlet fever, measles and whooping-cough, suggests the application of certain methods, used by Mr. Shirley Murphy in dealing with the altered age-incidence of diphtheria, to the task of ascertaining whether annual variations in age-incidence accompany variations in prevalence.

In so far as mortality can be regarded as an index to prevalence, the London records of deaths for a long series of years are available for this purpose. With a view to determining whether any alterations in age-incidence accompany the variations in London mortality, a series of diagrams has been constructed by finding the proportion borne by the

number of persons attacked at different ages in successive years to the number attacked at all ages during those years, this latter number being taken as 1000. The proportions so obtained have been set out in the form of percentage variations above or below a mean. Thus, in the case of small-pox in London between 1847 and 1881, the diagram* shows the extent of such variation above and below the mean in the case of the age periods, 0 to 1, 1 to 2, 2 to 3, 3 to 4, 4 to 5, 5 to 10, and 10 and upwards. The variations in age-incidence for each of these age periods may then be compared with the annual variations in the mortality from small-pox at all ages. It will be seen that, in the case of the age period 0 to 1, there is a tendency for increased incidence of the disease upon that particular age period, to manifest itself during the years when the disease is not specially destructive; and, on the other hand, during years of increased mortality the incidence upon the age period 0 to 1, is apt, generally speaking, to be comparatively small. In the case of the age period 1 to 2, a similar tendency is less clearly traceable, and in the age period 2 to 3, there is a suggestion of a reversal of the relationship indicated; thus in the years 1866-67, years of increased small-pox mortality, the incidence upon the age period 2 to 3, was also increased. The indications derivable from the age periods 3 to 4 and 4 to 5 are of a less definite character. In the age period 5 to 10, however, there is a fairly close correspondence—particularly noticeable in the earlier years of the series under consideration—between the years of increased small-pox mortality and the years in which increased incidence upon the age period in question is manifested. As regards the age period 10 and upwards, there is but little evidence of a kind similar to that afforded by the age period 5 to 10. The diagram, it will be noted, shows, of course, the striking contrast between the progressive diminution, when the whole series of years under consideration is passed in review, in the incidence of the disease upon the earlier age periods, and the striking increase in its incidence upon persons above ten years of age. There is, however, it may be stated, also evidence that, while the charts for some of the age periods tend to show increase and diminution in correspondence with increase and diminution of general small-pox mortality, the charts for other age periods show diminu-

* Reproduced in the upper portion of the diagram annexed to this paper.

tion and increase in correspondence with increase and diminution in the mortality at all ages.

In the case of scarlet fever,* during the years 1859 to 1893, the chart for the age period 0 to 1 shows several correspondences between a high rate of incidence upon the age period and a comparatively low rate of mortality at all ages from scarlet fever on the one hand, and a low rate of incidence and a comparatively high rate of mortality at all ages on the other. In the age period 1 to 2 the indications are less marked, but still tend to be to the same effect. In the age periods 2 to 3 and 3 to 4 there is still less correspondence to be noted. In the age periods 4 to 5 and 5 to 10, however, there are clear indications, particularly marked in the earlier years of the series under consideration, of a correspondence between a high rate of incidence upon these two age periods and a high degree of mortality at all ages on the one hand, and a low rate of incidence on the particular age periods, and a low rate of mortality at all ages on the other. The indications afforded by the chart for the period 10 and upwards are of a conflicting character.

In the case of measles, the indications are very obscure. There is, perhaps, some slight suggestion of a direct correspondence between the variations in incidence upon the age period 1 to 2, and the variations in measles mortality at all ages.

In all these charts the variations in age-incidence have been gauged by finding the proportion of deaths at each particular age period, deaths at all ages being taken as 1000. It might be suggested, however, that inasmuch as the higher age periods contain a larger proportion of insusceptible persons, a more precise comparison could be made by using deaths affecting the age period 0 to 2, which contains few persons who have been attacked, as a standard, and finding the proportion of deaths at each particular age period, those at the age period 0 to 2 being taken as 100. With the object of illustrating this, charts have been prepared by using the proportions borne by the deaths at the higher age periods to the deaths at the age period 0 to 2. In the case of small-pox, these proportions are shown† for the age periods 2 to 5, 5 to 10, 10 to 25, and 25 and upwards. The direct correspondence between the chart for the age period 2 to 5 and the chart immediately above it showing variations in the number of deaths

* See the upper portion of the diagram annexed to this paper.
† See the lower portion of the diagram annexed to this paper.

at all ages is seen to be fairly close, and somewhat similar indications are afforded by the chart for the age period 5 to 10. The chart of the age period 10 to 25 shows some correspondences in the later years under consideration. In the case of scarlet fever, the diagram shows* the proportions of deaths, at age periods 4 to 10, 10 to 15, and 15 and upwards, compared with the deaths at the period 0 to 2 taken as 100. Here, in the age period 4 to 10, the correspondence with the chart immediately above, showing variation in the number of deaths from the disease at all ages, is particularly close. For the period 10 to 15 a similar phenomenon is observed, though irregularities are marked, while irregularities are more pronounced than correspondences in the period 15 and upwards. In the case of measles, the age period 0 to 2 includes, it must be noted, the ages specially attacked; and the deaths at ages 0 to 1 and 1 to 2 appear to maintain fairly constant proportions to the total deaths at the period 0 to 2; but it will be seen that the chart showing the proportion of deaths at the ages 2 and upwards to deaths at 0 to 2, shows some indication of an inverse relationship to the variations in the deaths at all ages.

It will be observed, on the whole, that indications of the kind discernible in the case of small-pox and scarlet fever are but little marked in the case of measles. The figures of whooping-cough mortality have been investigated with a like negative result. It is to be noted, however, with regard to the material to hand in the case of measles and whooping-cough, that a division into age periods, 0 to 1, 1 to 2, etc., is not the one which would be naturally chosen in dealing with these diseases, were it possible to make a selection of age periods to be compared. Dr. Whitelegge has shown that the biennial rhythm of measles is only obscurely indicated, in the case of Nottingham between 1870 and 1890, when the yearly figures are consulted, but that it is apparent enough when quarterly rates are dealt with. In a similar manner it would perhaps be anticipated that any variations in age-incidence which might be observable, in the case of measles and whooping-cough, were it possible to deal with age periods 0 to 3 months, 3 to 6 months, etc., would be obscured when yearly periods come to be considered. In other words, the alterations from an inverse to a direct relationship between variations in age-incidence at particular age periods, and variations in mortality at all ages, which in the case of

* See the lower portion of the diagram annexed to this paper.

small-pox and scarlet fever are developed when ages from birth up to ten or more years of age are passed in review, may, perhaps, in the case of measles and whooping-cough, have already attained their development in the periods comprising only the first year or two of life. It would be a matter of great interest in this connection were it possible to study the variations of age-incidence in the Swedish population attacked by measles and whooping-cough, during the years when those diseases were displaying the unusually long cycles depicted in Dr. Ransome's diagrams.

Dr. Ransome attributed the length of the cycles in question to sparsity of population. In view of the fact of the sparsity of population in Sweden, it may be assumed that the Swedish figures would, taken as a whole, show in comparison with London figures a somewhat less marked incidence upon the earlier ages and slightly greater incidence upon the later ages: This assumption, it may be observed, is borne out by comparison between mortality rates at different age periods in London and in thickly-populated counties, and the corresponding rates in the more sparsely-populated counties of England. These differences, again, may be exhibited in a more exaggerated form by comparing the proportion of deaths at different age periods in sparsely-populated registration districts with those obtaining in London. Thus, taking fourteen sparsely-populated districts, I find that during a period of forty years the deaths from measles at ages 0 to 5 constituted only 78 per cent. of the total deaths, whereas in London such deaths constituted between 93 and 94 per cent. of the total deaths. These different mortality rates represent still greater differences in prevalence at the higher as compared with the lower ages. The question, however, which it would be interesting to decide is, whether the variations of measles mortality in Sweden, with their long waves, show variations of age-incidence more clearly than the short-waved London variations do; and, again, whether in London, in the event of its being possible to deal with quarterly age periods, similar variations in age-incidence would be brought to light by the process of artificially enlarging or magnifying, so to speak, the short (eighteen months to two years) intervals, and so placing them on a more equal footing with the longer periods of Sweden.

In the case of scarlet fever, and in that of small-pox, the indications afforded are of a more positive character. With regard to the age groups affected, it will be seen that in scarlet fever the ages which especially suffer at times of

increased destructiveness of the disease are those from 4 to 10. Of course the group 5 to 10 cannot be split up, and it may be that ages 4 to 7, or 4 to 8, would give more definite results than the whole group 4 to 10 does. On the other hand, it may be remembered that the age group 10 to 15 shows some indication of varying in the same manner as the preceding age group.

In small-pox the groups of ages which specially suffer during the cycles of prevalence of "the fifties" include younger persons than the corresponding group in scarlet fever. In small-pox, during "the seventies", higher age periods show a tendency to suffer from increased incidence of the disease during times when the general mortality is high.

The question now arises as to the significance of these phenomena. Are the alterations in age-incidence merely the effect of the variations in prevalence; or are they to be regarded as indicating variations in the amount of susceptible material in the population, which themselves have influence in producing variations in prevalence? It would, of course, follow that, given the existence of cyclical variations in prevalence, due, it may be, to variations in quality of virus (or influenced, in the case of scarlet fever, by some such cause as Dr. Longstaff has referred to in his observations with respect to the relationship between rainfall and scarlatinal mortality), there must result variations in the age-incidence observed in successive years. Thus, after a wave of prevalence has passed over a community, the proportion borne by the number of susceptible persons at higher ages to the number of susceptible persons among those who have been recently added to the community, must be comparatively small. This consideration would serve to explain why it is that the incidence upon the age period 0 to 1 should be high in a year of low (following upon a period of years of high) prevalence. How far such considerations are capable of accounting for the alterations in age-incidence which accompany alterations in the extent to which the general destructiveness of the disease is manifested, it is difficult to say. It must, of course, be borne in mind, on the other hand that, assuming that the mechanical accumulation of susceptible units plays an important part in determining variations of prevalence, and assuming that this factor produces variations in the extent of incidence upon persons belonging to different age periods at different phases of an epidemic cycle, this consideration would in itself serve to explain some variation in case mortality during the development

of a cycle of prevalence. In the case of the seasonal variations of scarlet fever, this question of the influence upon case mortality of age distribution has been investigated by Mr. Shirley Murphy, who finds that the lowered case mortality during the London autumnal prevalence of the disease, is not explicable as resulting from altered age-incidence, and possibly a similar analysis of the variations in case mortality in successive years, shown by hospital statistics of scarlet fever, would yield a like result.

There is a further question which arises, notably in the case of small-pox, as to the part played by mere mechanical accumulation of susceptible units in helping to bring about major prevalences of disease. It has been seen that the variations in age-incidence in the case of the early age periods, which are marked in "the fifties", become less marked in succeeding years. There is a suggestion in the small-pox chart that ages ten to twenty-five take up, in "the seventies", the *rôle* of showing an increased proportion of attacks as compared with attacks at all ages. In this connection, reference may be made to the demonstration given in the report of the Royal Commission on Vaccination, of the manner in which succeeding age periods take up an increasing proportion of the total mortality in consecutive quinquennia subsequent to the time when vaccination was made compulsory.

If it is to be assumed that successive accumulations of persons whose protection by vaccination had become enfeebled by the lapse of years have played a part in determining smaller cycles of prevalence, it is conceivable that the greater prevalence of 1871 may be, to some extent at any rate, explained by the altered circumstances brought about by the upgrowth of a generation for the most part protected from small-pox in infancy, but in the majority of whose units that protection, having become enfeebled, had not been renewed.

Another question which may be thought of in connection with variations in age-incidence at the different phases of cycles of prevalence, is that as to the ages which particular diseases are specially liable to attack. Thus, recurring to Dr. Ransome's three hypotheses, it might be suggested with regard to the "age theory", which, in its simple form and when considered by itself, Dr. Ransome held to be incapable of explaining recurrence in cycles of years, that this factor may have influence in modifying the mode of action of the principle that a certain density of population

at susceptible ages is necessary before a disease can spread with the vigour of an epidemic. In other words, if certain ages are specially liable to suffer from a particular disease, this fact must have influence in determining periodicity. On the other hand, periodicity undoubtedly affects the ages attacked; indeed, Dr. McVail writes, "the periodicity of small-pox is the governing factor in its age distribution"; and, on this view of the matter, the question—what age is most susceptible to attack by a particular disease?—may be held to be bound up with the question as to the periodicity which that disease has established for itself in the community under consideration. The facts deducible from the London mortality figures, which may have a bearing upon these questions, are those which relate to differences, observed in the diseases considered, with respect to the age periods which show an increased proportion of attacks in correspondence with increased general prevalence.

In conclusion, it may be remarked that the variations in age-incidence to which attention has been directed have been manifested in records of mortality; perhaps clearer indications would be forthcoming if it were possible to make similar inquiry with regard to attacks of disease. The London notifications scarcely afford as yet, however, sufficient material for such an investigation. Again, it would be interesting to have the opportunity of criticising impressions arrived at from a study of the London figures in the light of the figures relating to other communities.

THE REPORT OF THE ROYAL COMMISSION ON VACCINATION:

A REVIEW OF THE DISSENTIENTS' STATEMENT.

By JOHN C. McVAIL, M.D.

(*Read: February 19th*, 1897.)

The Report of the Royal Commission on Vaccination is signed by Lord Herschell, the Chairman; Judge Meadows White, Q.C.; Mr. John S. Dugdale, Q.C.; Sir Edwin Galsworthy, Chairman of the Metropolitan Asylums Board; Sir Charles Dalrymple, M.P.; Mr. Samuel Whitbread, ex-M.P.; and Mr. John A. Bright, ex-M.P. It is also signed by the following members of the medical profession: Sir James Paget, Bart.; Sir W. Guyer Hunter, ex-M.P.; Prof. Michael Foster; and Mr. Jonathan Hutchinson. These include all but two of the Commissioners.

The Commission heard 187 witnesses, of whom the great majority were put forward to represent the case for anti-vaccination.

The above-named members are unanimous in conclusions which may be summarised as follows:—

The Value of Vaccination and Re-vaccination.

1. That vaccination diminishes liability to attack by small-pox.

2. That when attack does occur in vaccinated individuals, their vaccination renders the disease milder and less fatal.

3. That protection against attack is greatest during the years immediately following vaccination, and that the period of highest protection may perhaps be fairly stated as covering nine or ten years.

4. That after the period of highest protective potency, the efficacy of vaccination against attack is considerable in the next quinquennium, and possibly never altogether ceases, but that it rapidly diminishes.

5. That the power of vaccination to modify attack outlasts its power to protect against attack, and that its efficacy in this respect is still very considerable, even during the later periods of life.

6. That re-vaccination restores protection, but that this

restored protection again diminishes, and should be renewed if it be desired to ensure the highest degree of protection which vaccination can give.

7. That vaccination is beneficial according to the thoroughness with which it is performed. **The Safety of Vaccination.**

Regarding alleged evil results of vaccination, it is necessary to note the means taken by the Commission to ascertain and inquire into cases:—

"Section 405. Since the 1st of June 1889 we have, from time to time, been informed from various sources of cases in which death or non-fatal injury has been alleged or suggested to have been caused by, or otherwise connected with, vaccination, with a view to their investigation; and since the 14th of February 1891 the Local Government Board have immediately informed us of all such cases brought to their notice. In March 1892 the Home Office addressed a circular letter to coroners throughout England and Wales, requesting that in all cases where they received information that the death of any person, on whose body they proposed to hold an inquest, had been alleged to have been caused by, or to have had any connection with, vaccination, they would communicate immediately with the Commission."

In Section 407 they write thus:—

"We have not any means of ascertaining in what number of cases some other disease has supervened on vaccination as a consequence of it, without producing a fatal result. We are able, however, to form some judgment upon this point by observing the number of non-fatal cases to which our attention has been called. We do not mean to suggest that we have been informed of all cases of this nature which have occurred during the last six years. There have very likely been many cases which have not come to our knowledge, where the inflammation set up has been more than usual, and some where a slight attack of erysipelas has resulted. But when we consider that the fact that we were engaged upon this inquiry has been thoroughly well known, and that active organizations and zealous individuals were at work searching out cases in which the results of vaccination had been abnormal, with a view to bring them under our notice, and that some of those which we were asked to investigate turned out to be of a trifling and unsubstantial nature, we think we are able to form a fairly accurate estimate of the amount of injury which can be plausibly attributed to vaccination. A consideration of all the circumstances has led us to the

conclusion that, as regards the non-fatal cases with which we are now dealing, serious injury cannot have resulted in any considerable number of cases."

Inquiries into cases of supposed injury were conducted, not by the Medical Department of the Local Government Board, but by medical men appointed for the purpose by the Commission. The general conclusion, as stated in Section 434, is as follows:—

"A careful examination of the facts which have been brought under our notice has enabled us to arrive at the conclusion that, although some of the dangers said to attend vaccination are undoubtedly real and not inconsiderable in gross amount, yet when considered in relation to the extent of vaccination work done they are insignificant. There is further reason to believe that they are diminishing under the better precautions of the present day, and, with the addition of the further precautions which experience suggests, will do so still more in the future."

The Indispensability of Vaccination.

Another question is whether isolation in hospitals could be relied on in place of vaccination, and here the Commissioners report (Section 503):—

"We can see nothing then to warrant the conclusion that in this country vaccination might safely be abandoned, and replaced by a system of isolation. If such a change were made in our method of dealing with small-pox, and that which had been substituted for vaccination proved ineffectual to prevent the spread of the disease (it is not suggested that it could diminish its severity in those attacked), it is impossible to contemplate the consequences without dismay.

"To avoid misunderstanding, it may be well to repeat that we are very far from underrating the value of a system of isolation. We have already dwelt upon its importance. But what it can accomplish as an auxiliary to vaccination is one thing; whether it can be relied on in its stead is quite another thing."

On the above points the Signatories are unanimous.

Enforcement of Vaccination.

As regards the particular degree of enforcement which should be maintained by law, there is some difference of opinion: Mr. Whitbread and Mr. Bright believing that even modified compulsion is unnecessary and impracticable, while Sir Guyer Hunter and Mr. Jonathan Hutchinson believe that there should be compulsion with regard to both primary vaccination and re-vaccination. The other signatories take a middle position.

THE DISSENTIENTS' STATEMENT.

Two members of the Commission dissent from the Report as a whole, and from the conclusions above specified. These members are Dr. W. J. Collins, formerly a Vice-President of the London Society for the Abolition of Compulsory Vaccination, and Mr. J. A. Picton, lately Member of Parliament for Leicester. Their Grounds for Dissent are contained in a Statement issued along with the Commission's Report. From the Statement it appears that the two dissentients bore a share in the preparation of many parts of the Report, but that they had no share in some other parts. In preparing their own Statement they seem to have had, as was convenient, the Report before them, including parts of it in whose preparation they had not shared, and some sections of their Statement may be looked on as a criticism of the Report. It is very unfortunate that those portions of the Report in which they did take a share have not been specified, as the specification might have given a fuller view of the opinions of the two dissentients regarding vaccination than is obtainable in the Statement itself. Whether any of these portions were modified by the Commission, so as if possible to meet the views of Dr. Collins and Mr. Picton, who after all refrained from signing the document, can only be surmised; but this is of less consequence, as the conclusions stated in the Report regarding (*a*) the value of vaccination as a protection against small-pox; (*b*) the impossibility of protecting against small-pox without vaccination; and (*c*) the practical safety of vaccination, are sufficiently strong and explicit as they stand, however much more forcibly they might otherwise have been stated.

It is the purpose of the present paper to traverse most of the more important criticisms made by the dissentients. Their attack on vaccination is skilfully conceived and well delivered. Indeed, in perusing the Statement, the reader who is acquainted with the literature of the subject is likely soon to arrive at the opinion that no more ingenious attempt has ever been made to discredit vaccination. It is not their fault, but their misfortune, that the evidence for vaccination is so strong as to resist all efforts to break it down. The Statement consists of 303 sections, and in the course of the following notes it will be convenient to refer to these rather than to pages.

It begins as follows :—

> "We entirely agree with the Report of our colleagues in so far as it shows the great change of professional and scientific opinion since vaccination first engaged the attention of the Legislature, and since the passing of the first compulsory Act in 1853. We hold with them that the prophylactic power of vaccination has been at least exaggerated, and that dangers incidental to the practice, though at one time denied, are undoubtedly real and not inconsiderable in gross amount."

The Commission assumes, not unnaturally, that with regard to vaccination, as with regard to most other things, much has been learned and opinions on various points have been more or less altered, since the beginning of the century. That Jenner and his contemporaries were wrong in believing that either an attack of small-pox, or inoculated small-pox, or vaccination, could certainly confer life-long protection against future small-pox, is one of the essentials of the modern doctrine of vaccination; and in "holding" that this is so, the dissentients only express their agreement with the opinions which have been long maintained by the medical profession, and to which it is earnestly desired that practical effect should now be given by the Legislature, by the introduction of a national system of re-vaccination.

A Question of Degree.

> "We desire also," say the two dissentients, "to give reasons for thinking that other more effective and practicable (as well as less objectionable) modes of stamping out small-pox or protecting communities from its introduction are available."

This is the first of a series of passages in the Statement which show that the dissentients are of opinion that vaccination *is* an agency of use for stamping out small-pox and for protecting against its introduction, their dissent so far consisting in "thinking"* that other means are better than vaccination. In 1889 Mr. Picton declared in the *Contemporary Review* that he was "rapidly approaching" the conclusion "that vaccination is of no use." It is evident

* The fact that the Commission prefix their seven conclusions by the judicial phrase, "We think", has been largely advertised by opponents of vaccination, even by a lawyer like Mr. Hopwood, Q.C. Taking their own reading of the expression, they should observe that the dissentients also have only got the length of "thinking" that other measures are "more effective and practicable" than vaccination.

that his seven years' study of the subject as a Commissioner has not succeeded in bringing him to the conclusion to which he had then so nearly arrived. He does not now say that vaccination is of no use, but only that he thinks there are other "more effective and practicable (as well as less objectionable) modes" of dealing with the disease. At once, therefore, the dissent is narrowed down to a question of degree, and it is important to bear in mind that this is all the length the former Vice-President of the London Society and the ex-M.P. for Leicester have been able to go.

The Standpoint of the Commission.

"We venture to think that the Report of our colleagues, in the preparation of many portions of which we have borne our part, has approached the consideration of the behaviour of small-pox and the means of preventing it, too exclusively from the standpoint of vaccination; and that too little attention has consequently been accorded to sanitary organisation, prompt notification and isolation, measures of disinfection and cleanliness, and healthy conditions of living, which we believe to be of the first importance in preventing and controlling outbreaks of small-pox."

Here again the words "too exclusively" and "too little" show that the difference between the Commission and the dissentients is one of degree; but the standpoint from which it was the duty of the Commission to view the subject can best be judged of by the questions originally submitted to the Commission by Her Majesty. These were:—

1. The effect of vaccination in reducing the prevalence of, and mortality from, small-pox.

2. What means, other than vaccination, can be used for diminishing the prevalence of small-pox; and how far such means could be relied on in place of vaccination.

3. The objections made to vaccination on the ground of injurious effects alleged to result therefrom; and the nature and extent of any injurious effects which do, in fact, so result.

4. Whether any, and if so what, means should be adopted for preventing or lessening the ill effects, if any, resulting from vaccination; and whether, and if so by what means, vaccination by animal vaccine should be further facilitated as a part of public vaccination.

5. Whether any alterations should be made in the arrangements and proceedings for securing the performance

of vaccination; and, in particular, in the provisions of the Vaccination Acts with respect to prosecutions for non-compliance with the law.

Looking to these questions, it was obviously the duty of the Commission to consider primarily the question of vaccination; and it is needless to discuss whether the dissentients, instead of approaching the subject from the standpoint of vaccination, have preferred to do so from the standpoint of anti-vaccination.

The Antiquity of Small-Pox.

Sections 2 and 3 illustrate the curious tendency on the part of opponents of vaccination to limit the antiquity of small-pox. Some have held that the disease was known in ancient Greece and Rome; others that this is doubtful; and others that it was unknown. The dissentients adopt the last opinion, and with regard to the well-known work on small-pox by Rhazes of Bagdad, while they point out that it was written in the tenth century they do not state that it contains various references to earlier writers on small-pox whose works are not now extant. So also as concerns the disease in Europe and in England: they prefer to ignore the earliest evidence, and to accept extreme views as to the comparatively recent periods in which the disease has largely prevailed. The question is of little practical moment, but Dr. Collins and Mr. Picton seem to attach much importance to it, and in Section 6 they write:

> "During the seventeenth century small-pox became more prevalent in Western Europe, especially in large towns and trading ports".

This assertion is unproved. Statistical records for early centuries are scanty: but it is rash to assume that because tables of figures are more common for recent centuries, the disease itself was less prevalent previously. As Hirsch says: "the materials available to us are not even in a measure adequate for drawing up a proper history of the pestilence in past centuries. It was not in its rarity, but as Häser justly remarks, actually its everyday occurrence, that weakened the interest of epidemiographists towards it." There is no good reason to assume that if statistics had existed for European populations earlier than those for Geneva (which begin in 1580), the disease would have been shown to be less prevalent.

Regarding infectious diseases in general, the accepted

view is that each case depends on a previous case, and that there is no *de novo* origin. This being so, it is obvious that isolated communities, like the Faroe Islands, are much less liable to infection by small-pox or measles, or other similar disease, than places in closer or more constant touch with the rest of the world; but this does not give much help to the case of the dissentients with regard to the United Kingdom. Even in a country so isolated as Iceland, the annals record that small-pox raged frequently and severely from 1241-2 onwards.

Section 7:—

Plague and Small-pox.

"In London the ravages of the Plague, until its extinction in 1680, appear to have eclipsed the lesser evils of the small-pox, and to have absorbed whatever of organised effort was available on the part of those responsible for the public health."

As to the Plague there is no evidence that organised effort had much to do with its extinction, but concerning the relative importance of plague and small-pox, Macaulay writes, in a passage which has been frequently quoted:—

"That disease over which science has since achieved a succession of glorious and beneficent victories, was then* the most terrible of all the ministers of death. The havoc of the Plague had been far more rapid; but the Plague had visited our shores only once or twice within living memory; and the small-pox was always present, filling the churchyards with corpses, tormenting with constant fears all whom it had not yet stricken, leaving on those whose lives it spared the hideous traces of its power, turning the babe into a changeling at which the mother shuddered, and making the eyes and cheeks of the betrothed maiden objects of horror to her lover."

The London Bills of Mortality.

Continuing the same line of argument, Section 8 begins by referring to the London Bills of Mortality, and proceeds to give a table of annual death-rates in London per 100,000 living in two periods of the seventeenth century. It would have been convenient for the general reader had it been pointed out that the table is not given in the Bills of Mortality, and that as the Bills give no statement of population, death-rates calculated on population are unreliable. Besides, the earlier of the two periods given

* Death of Queen Mary, wife of William III.

in the Table is so short—six years only—that taken by itself it can yield no satisfactory averages.

In Section 10 the assertion is made that:—

> "In 1710, for the first time since the Bills of Mortality had been compiled, more than 3,000 deaths were ascribed to small-pox in London, or 127 per 1,000 deaths from all causes."

This is partly incorrect. The Bills for small-pox begin in 1629. Some years are wanting, but in 1634, the deaths from small-pox were 130 per 1,000 deaths from all causes. The population within the Bills in 1634 was, of course, much less than in 1710, so that the total deaths, both from small-pox and from all causes, were less. So far as it is not incorrect, the statement is apt to mislead, for in 1681, or nearly thirty years before 1710, and with a smaller population, the total small-pox deaths within the Bills were no less than 2,982. It should be understood also that in connection with the Bills only burials in recognised places of sepulture are included, and not all deaths.

The Contagion of Small-Pox.

In Sections 4 and 5 the dissentients say:—

Sydenham's contemporaries.

> "Sydenham, like his contemporaries, did not attribute the propagation of small-pox to contagion, but to what he termed 'the epidemic constitution of the atmosphere', due to 'certain hidden and inexplicable changes within the bowels of the earth'."
>
> "It was reserved for Boerhaave, of Leyden (1668-1738), to proclaim the view, now generally accepted, that small-pox arises only from contagion."

This is the first indication of an argument that the prevalence of small-pox in past centuries was largely due to the want of knowledge of its infectious nature, and that if this had been known the disease would probably have been checked long ago by means of isolation. To begin with, however, the statement regarding Sydenham's contemporaries is incorrect. Both Willis and Martin Lister, who were among his most distinguished contemporaries, fully recognised the contagious nature of small-pox, and so also did Riverius, who preceded Sydenham,* and whose works were widely published.

* Riverius (born 1589) wrote:—"Postquam verò Aethiops quidam, eò delatus, variolis pestilentibus corruptus est: communicata per contagium maligna et

MEAD ON THE PLAGUE.

> "The prevalence of the disease led to many speculations as to possible means of deliverance from it. The orthodox teaching of propagation by 'epidemic constitution of the atmosphere' was not calculated to inspire sanitary precautions, or the separation of the sick from the whole. Mead's work on the prevention of contagions, primarily directed against a threatened invasion of plague, was not written until 1720. On the other hand, there were reports from the Levant, where small-pox had long been endemic, that by a method of 'engrafting' the disease artificially, it might be robbed of its terrors." (Section 10.)

Any suggestion that if small-pox inoculation (or engrafting) had not been introduced from about the year 1721, Mead's directions for prevention of the Plague would or might have been applied to prevention of small-pox, will hardly bear examination. The last important visitation of the Plague had been in the year 1665, and its terrible nature had caused it to be well remembered, so that there was much anxiety regarding its threatened return in 1720. There was, however, no Plague in England. Mead's proposals amounted to quarantine in the literal sense—forty days' detention of vessels or persons coming from infected countries and under suspicion. The Plague was, indeed, looked on as a foreign disease. A Foreign Disease.

He advised hospitals, built on islands if possible, destruction of clothing, even burning of ships. In the event of the Plague finding entrance into a country, he recommended more hospitals, burning of infected clothing and

venenata qualitate, tam immaniter grassari coepit affectus ut magna Indorum pars de media sublata fuerit."—*Opera*, Frankfort, 1674, p. 548.

Thos. Willis (born 1622) in *De Febribus*, cap. xv, says:—"... etiam aliqui contagium facile suscipiunt; alii vero sine periculo saepe cum aegris conversantur." Again:—"Contagio hunc morbum in alios serpere, latesque grassari, quotidiana experientia manifestum est; scilicet a corpore infecto continuo decedunt effluvia, quae, ab aliis corporibus suscepta, statim, instar veneni, cúm sanguine permentescunt, et semina ejusdem affectûs latentia, ipsisque homogenea suscitant, et in hujus morbi ideam disponunt: nec solum contactu, sed ad distans miasma communicatur. Qui intra ejusdem aedes, aut in aegrotantium vicinia degunt, facile labem suscipiunt; necnon et vestium interventu fometur, longius dissipatur, et ad remota transferetur loca."

Martin Lister (born about 1638) writes thus in *De Variolis* (1697):—"Illud quidem verissimum est (a primis hujus autoribus traditum quod ipsi etiam quotidie animadvertimus) variolas esse de summe contagiosis aegrotationibus."

Sydenham was born 1624.

houses, sanitary inspection, cleansing, attention to personal health, avoidance of infection, prohibition of public meetings, and the forming of *cordons* round infected towns. Mead's scheme is interesting, and much of it is excellent. A good deal of it, however, was by no means new.* That detracts little from the credit due to Mead, but is of consequence in connection with the view (if such a view is really intended) that Mead's proposals dropped into oblivion owing to the adoption of small-pox inoculation. Some of Mead's regulations are admirably thought out, and he was among the first to insist that if the Plague did come, the inmates of infected houses should not be shut up with the infection. Even with regard to this, however, Simon notes that, so early as 1629, "at first they had shut up the sick in their houses, but on further consideration had thought it better that the house should be shut up and the inmates sent to the pest-house"—though this proposal was probably very imperfectly carried out, and we read later on of the continuance of the horrible practice.

The question for us is: Was there the slightest chance of such a system as Mead's being introduced with regard to small-pox, even if small-pox inoculation had never been heard of? Let it be noted that Mead himself does not seem to have dreamt of it for small-pox. The measures refer to the Plague, a foreign disease which had been absent from the country for half a century, and attacked and slew people at all periods of life. Small-pox, however, was constantly present, was very widely spread, and was mainly a disease of children. In fact, plague and small-pox in Mead's time may, with regard to their prevalence and age-incidence, be roughly compared to cholera and measles in the present day. Like plague, cholera is looked on as a foreign disease destructive to adult life, while measles is always with us and is a disease of childhood. Nowadays, with regard to cholera, we have advanced so far as to adopt means more or less similar to those which Mead recommended for plague. With measles it is very different. No one, or almost no one, proposes to provide hospital accommodation for all cases of measles, and quarantine accommodation for exposed families; and even among the few local authorities which have made measles notifiable, some are pondering whether they should not give up their attempts to deal with it. So in 1720,

Small-pox Endemic.

* See Simon's *English Sanitary Institutions*, chap. vi.

small-pox was a children's disease everywhere prevalent, and not even Mead, who was probably the most far-seeing sanitarian of his time, mentions his scheme with regard to what he calls "the more common contagion of the small-pox". In the present day, hospital isolation is of much use in dealing with small-pox; but the fact that it is a possible agency in helping to check the spread of the disease is due to the previous fact that vaccination has made small-pox a comparatively rare malady, whose victims in a population are, therefore, sometimes so limited in numbers as to be able to be fenced off from their neighbours. The infection of scarlet fever is more easily controlled than that of small-pox; and here isolation is of use, even in the absence of any means like vaccination, though largely as a result of this absence scarlet fever remains many times more prevalent than small-pox.

Small-Pox Inoculation: Its Effect on Small-Pox Mortality.

The dissentients now enter (Section 13) on the subject of small-pox inoculation. *In limine* they indicate a doubt as to the truth of the view that, "in the case of many infectious diseases one survived attack affords a certain amount of protection against a second attack." Here, however, the general experience of mankind is too strong to be appealed from; and they turn to the other question as to whether a second attack of small-pox, when it does occur, is milder than a first attack. They state that—

The Fatality of Second Small-pox.

> "Experience, though of limited amount, appears to show that no mitigating influence is exerted by the first upon a second attack should it occur."

In support of this statement they refer to two answers given in the Minutes of Evidence, one of these being by the writer of this paper. That answer (Q. 24,937) is a very lengthy one, and cannot conveniently be given in full, but the first reference to the point is in a quotation from Bryce, who says "that these second attacks of small-pox have been in general more mild than the first attacks; and that the horn-pock and stone-pock, names known long before the days of vaccination, occurring after small-pox, are really to be considered as second attacks of that disease." Of course horn-pock and stone-pock were very mild maladies compared with small-pox, and as second

attacks of small-pox they would show much mitigation. The answer goes on to refer to various writers who speak of similar modifications of small-pox following on small-pox. Then it is pointed out that Dr. Seaton gives 167 cases of second small-pox with 36 deaths; that Marson mentions a fatality of 19 per cent.; and that the Epidemiological Society reported 200 cases of second small-pox with a fatality of 8.3 per cent.; and it is argued that the fatality from second small-pox is in reality low, but that this is not always obvious at first sight, partly because mild cases, as as to whose nature there might be doubt, would probably be excluded, and partly because second small-pox is likely to occur at a high range of ages when the mortality from normal and primary small-pox is high. The answer, therefore, to which they appeal does not seem to support the view expressed by the dissentients.

> "Notwithstanding the extensive practice of inoculation, or, as has been alleged, in consequence of it, small-pox continued throughout the eighteenth century to be endemic in London, and severely epidemic, often at frequent intervals, in many towns and villages in this country and abroad. During the latter half of the century, attention was called by many writers to the serious evil to society of partial and indiscriminate inoculation. It was shown that, whatever advantages might result to the inoculated by way of protection from attack, the practice had frequently been the means of introducing the disease into towns and villages that were previously free from it, and that it could only be worked at an intolerable cost of life."—Section 15.

It is unfortunate that no list of the "many writers" during the latter half of the century is given, and that no one of them is mentioned by name, though Dimsdale and Haygarth might both have been quoted in support, but only up to a certain point. The marginal reference is to the Papers of Sir John Simon in 1857, but Simon himself quotes from two writers in the beginning of the present century—Heberden in 1801, and Moore in his *History of Small-Pox* in 1815. The question is whether the practice in the last century did more good by protecting those inoculated than harm by spreading the disease among persons who did not choose to submit themselves to inoculation. Some writers, like Heberden and

Blane, held that more harm than good had been done. Others, like Gregory and Adams, and in Scotland many contributors to Sir John Sinclair's "Statistical Account" (1791-1799), held that the balance was decidedly on the side of good, and the Royal Commission has (Section 75) concluded that:

"There is no adequate evidence that inoculation did increase the mortality from small-pox. There was certainly, so far as the evidence goes, no such increase of small-pox, coincident in point of time with the increase of inoculation, as to justify the decrease of the latter being considered the main cause of the marked decline of the former. Nor is there sufficient even to show that it was a distinct subsidiary cause."

In Section 22 there is quoted a statement made in 1817 by Dr. Moore, in his *History of Vaccination,* who was an ardent supporter of vaccination as contrasted with inoculation, to the effect that, in the last century, want of care in preventing the spread of the disease from the inoculated "occasioned the loss of millions of lives." It is impossible that Dr. Collins and Mr. Picton can look on this as anything else than a wild exaggeration; and it would have been well had they added a comment to that effect, instead of giving the quotation and passing on. They declare themselves "satisfied that inoculation did tend to establish and spread the disease, and introduce it into places which would probably otherwise have remained free, and that in places where it was restricted a less mortality resulted." In the course of the evidence, however, the writer was asked the following questions by Dr. Collins:—

(*Q.* 25,876)—"Would not the question of its doing any amount of good by way of reducing the fatality in those who had been inoculated depend largely on the extent to which it had been practised?"—"To some extent it would." (*Q.* 25,877)—"So that its partial practice might be more deleterious than its universal practice?"—"Undoubtedly."

It would therefore appear that its partial practice or restriction in the early part of the present century may have done more to spread the disease than did its more general practice in the last thirty years of the eighteenth century, when, according to Gregory, who could only have been speaking very generally, its use was "almost universal".

In Section 24, it is argued that small-pox inoculation would probably have the effect of spreading the disease in isolated towns and villages "removed from the more

populous centres of human intercourse, and in which accordingly small-pox came rarely and epidemically." As a matter of fact, however, there is in Sir John Sinclair's "Statistical Account of Scotland," published in a series of volumes from 1791 to 1799, ample evidence that in rural populations small-pox inoculation did confer very great benefits; and that previous to inoculation there had been no such freedom irom the disease as is reported with regard to certain villages in Kent, whose immunity has been attributed to avoidance of inoculation.

De Haen, whose name is also mentioned (Section 27), founded his opinions on the London Bills of Mortality; and his want of acquaintance with the facts is such that he speaks of the years 1733 to 1755 as "famous for inoculation" in London; while he further purports to contrast the deaths from small-pox in England in two periods of thirty-eight years before and after the introduction of inoculation. Needless to say, no statistics exist on which to found such a contrast. The different lessons which figures may give when differently read is curiously illustrated in connection with Section 25. It is there pointed out that, in the first quarter of the eighteenth century, small-pox contributed 7.6 per cent. of the total deaths of London; that in the second quarter its contribution was also 7.6 per cent.; that in the third quarter it was 10.3 per cent., and in the fourth quarter 9.2 per cent.; and it is urged that this shows that the proportion of small-pox deaths to deaths from all causes was greater after the introduction of inoculation. On the other hand, Dr. Gregory, of the Small-Pox Hospital, taking cognisance of the historical fact that small-pox inoculation after its introduction in 1721 rapidly fell into desuetude, but revived towards the middle of the century, makes the following statement:—

Statistical Groupings.

"If, for instance, we divide the last ninety years of the eighteenth century into three periods, we shall find that the recorded deaths by small-pox were as follows:—1711-1740 (when there was no inoculation), 65,383; 1741-1770 (when inoculation was coming into general use), 63,308; 1771-1800 (when inoculation was almost universal), the deaths were only 57,268; so that, by this showing, inoculation diminished the mortality by 8,115 lives."

Regarding these figures, the dissentients cannot well urge that the existence of a small amount of inoculation previous to 1740 vitiates the argument: as in Section 25 they hold that, for various reasons—

"It would probably be difficult to conceive conditions more favourable to the spread and fatality of small-pox than those which obtained in London in the first three quarters of the last century."

In their statement on this question of the influence of inoculation, Dr. Collins and Mr. Picton confine themselves rigorously to writers who hold that small-pox was increased and not diminished by inoculation, and they make no reference to any of the very considerable amount of evidence to the opposite effect.

Haygarth's Plan to Exterminate the Small-pox.

We have already discussed the question whether Mead's proposals of 1720 for preventing the Plague might have been carried out with regard to small-pox, had not the practice of inoculation been introduced in 1721. With regard to vaccination, there is now put forward a somewhat similar suggestion on a basis of no greater validity: namely, that a scheme proposed by Dr. Haygarth of Chester in 1784, and again in 1793, "to exterminate the small-pox", might have been adopted had not the profession become occupied with the subject of vaccination after the publication of Jenner's first work in 1798.

"Some, like Haygarth, suggested the combination of general and systematic inoculation at stated intervals, with measures of isolation." (Section 16.)

"It was at this juncture that the value of the cow-pox as a protection against small-pox attracted attention". (Section 17.)*

A Healthy Town.

Much has been made of this line of argument, and it is necessary to scrutinise the facts. Haygarth was a physician in Chester, a town which he describes as of "almost incredible healthiness," notwithstanding the fact that in the year 1775 only 1,060 persons out of a population of 14,173 had never suffered from small-pox. Haygarth was an enthusiastic sanitarian, and all credit is due to him for his anxiety to stamp out small-pox. In considering the subject he had arrived at several opinions, which, if only they had been correct, would have greatly facilitated his object. Unlike Mead, who believed that the infection of plague could be conveyed by goods and especially by cloth goods, Haygarth persuaded himself that clothing could not convey the poison of small-pox unless it were directly smeared

Haygarth's Doctrine.

* See also Sections 221 and 238-40.

with matter from small-pox pustules. He further held that the poison of small-pox could not, as a rule, be conveyed through the air for a greater distance than 18 inches, and that a small-pox patient confined in one room in a house would not spread the disease into the next room, or into the passage, etc. It was, therefore, in his opinion, very easy to prevent small-pox from spreading, even among closely adjoining houses. Another of his opinions was that there was no risk of infection from a case of small-pox until the third or fourth day after the beginning of the eruption, or, in his own words, "till the eruption is completed." Holding these comfortable doctrines, he did not even then propose to stamp out small-pox by means of isolation alone. His plan was to combine isolation, or such isolation as according to his theories would be deemed sufficient, with general and periodical small-pox inoculation, and he formed in Chester a Society to work out the plan. The general inoculations were to be carried out in every second year, and any small-pox cases occurring in the interval were to be subjected to so-called isolation. The society seems to have gone on for four or five years, but when the time came for the third biennial inoculation, it was found that the people would have nothing to do with it, and in consequence the whole scheme broke down. Haygarth's first work on the subject was written in 1784 and the breakdown occurred very early. His most commendable enthusiasm, however, led him in 1793 to write another book, in which he suggested that his plans should be made matter for legislation and applied generally throughout the country, especially in the larger towns. The plans were not adopted. It was fortunate for the future of isolation that they were not adopted. To attempt to stamp out small-pox by a scheme based on such essential errors as his would have ended in bringing isolation into general contempt. Any assumption that it was the introduction of vaccination in 1798 which prevented the adoption of Haygarth's schemes, as set forth in 1784 and 1793, is gratuitous, but if such an assumption had sufficient basis, then Jenner and vaccination would have to be thanked and not blamed for preventing experiments in isolation which could have resulted only in failure.

By directing men's minds towards the subject of isolation, Haygarth's writings probably contributed not a little towards education in public health, and all credit is due to him for the perseverance with which he urged the adoption of his proposals; while the fact that so little was done to

put his system into practice prevented the good tendency of his teachings from being more than nullified by his blunders as to the fundamentals of any workable scheme. It is a curious illustration of the vitality of error that, within the last two or three years, Haygarth's writings are being used by the dissentients and others in support of foolish and visionary proposals for stamping out small-pox by isolation, independently of vaccination. In justice to Haygarth it must be borne in mind that he himself was guilty of no such folly, as his isolation proposals were only one half of a scheme of which the other half consisted in a system of general inoculation.

Faust, who is also referred to by the dissentients (Sections 16 and 240) in connection with proposals for isolation, had read Haygarth's writings, and, agreeing with him in some of his utterly mistaken theoretical views,* seems to have arrived at the conclusion that isolation alone was sufficient to prevent small-pox, without the aid of inoculation. Faust.

Cappel had translated Haygarth into German; and apparently also under the influence of Haygarth's false teaching as to the *modus* of the small-pox infection, he had, like Faust, formed the opinion that protection by inoculation was needless, and that isolation itself ought to suffice for preventing a disease which was conveyable for only 18 inches through the atmosphere, which could not be carried by clothing, and which did not begin to be infectious until several days after its eruption had appeared. If on the Continent it be the case that these proposals of Haygarth's as modified by Faust and Cappel were not carried into practice owing to the interest which was developed in vaccination, then once more vaccination is to be thanked for having prevented isolation falling into disgrace and disrepute by being submitted to an utterly fallacious test. Cappel.

With regard to isolation proposals, the dissentients also quote the name of Rast (Section 16); but on verifying the references given by them I cannot find any mention of Rast, nor does his name seem to be given in the index to the volume of evidence in question. Again, at the beginning of Section 27, mention is made of Rast, and two marginal references are given, namely to Q. 10,515 in the Minutes of Evidence and to page 66 of the appendix to vol. 1 of the Rast.

* He quotes a statement from Haygarth to the effect that when small-pox breaks out in a slave-ship, the separation of the first case prevents the spread of the disease; and he declares "that small-pox is not propagated by the air but by contact, or by approaching its atmosphere, which is fully confirmed by experiment".

Minutes of Evidence. Once more, however, Rast and his opinions elude the inquirer, for his name is not mentioned either in Q. 10,515 or in the appendix referred to.

If anything further were required to show that vaccination cannot be blamed for having hindered any attempt at genuine isolation, it is to be found in the fact that in 1801 Haygarth advocated practically similar measures of isolation for the prevention of fevers. Here, of course, there was no scheme corresponding to vaccination which could interfere with the carrying out of his proposals. Yet it does not appear that anything more definite in the way of isolation followed with regard to fevers than with regard to small-pox.

In Section 30 speaking of the beginning of this century, the dissentients mention—

> "... efforts, which were for the first time made to restrict the spread of small-pox—by efforts directed against contagion."

Possibly Haygarth's writings are here referred to, but if so we have seen that they did not result in "efforts," excepting for a few years in Chester, about or before the beginning of the last vicennium of last century.

It may be noted in passing, that in the course of a quotation from Haygarth the dissentients italicise a part of it, emphasing the benefits which—

Prevention by compulsion.

> "*might reasonably be expected from the united, systematic, and concerted regulations of the whole island, aided and strengthened by legal premiums and punishments.*"

This is the first indication of the curious position which Dr. Collins and Mr. Picton ultimately take up: that while compulsory vaccination is an unwarrantable interference with individual liberty, removal to hospital ought to be compulsory.

Small-Pox, "Fevers" and Sanitation.

In its broadest sense, the term Sanitation includes all reasonable means available to humanity for conserving the public health, such as vaccination and isolation, but it is often convenient to limit its use, so as to refer only to measures for permanently improving the surroundings of the individual, by providing him with pure air, water and food, good house accommodation, and so forth. The word

is here used in that restricted sense, and sometimes it will have to be still further limited.

In Sections 17-19 the question of the influence of sanitation on the prevalence of small-pox is again raised. Figures are quoted for London to show that small-pox deaths diminished during the last twenty years of the eighteenth century. The diminution has with equal reason been adduced to prove that inoculation—which Dr. Gregory declares to have been at that time more prevalent than ever before—had caused a reduction in small-pox mortality. A table is given (Section 18) to show that the deaths, not only from small-pox but from all causes, and from "fever," have progressively dimi- "Fever. nished in London. Taking the figures for what they are worth (and for previous centuries they purport to show death-rates on a population which can be no more than roughly guessed at), it is to be noted that while the deaths from all causes have diminished, those from small-pox have, since Jenner's time, diminished very much more rapidly. In the successive periods since vaccination was introduced (beginning 1801-1810) the fraction contributed by small-pox to the total mortality has fallen somewhat as follows:—$\frac{1}{15}$th, $\frac{1}{40}$th, $\frac{1}{50}$th, $\frac{1}{60}$th and $\frac{1}{120}$th. Fevers, however, have also been reduced at a much greater rate than the deaths from all causes, and the dissentients attach much importance to this fact, apparently to support the suggestion that small-pox also might have been reduced without the aid of vaccination. But it is hopeless to endeavour to establish any useful analogy between the diminution in the registration of deaths from "fever" and deaths from small-pox. As is pointed out in the Report of the Commission, the nomenclature of fevers has greatly altered, and diseases which were at one time included under this term are now classified differently. In defence of their position, the dissentients quote from Mr. Simon to show that Dr. Greenhow and Dr. Farr, in preparing the earlier figures of the Table quoted in the Statement (Section 18), took pains to include as fever all diseases which might be embraced in the old application of the word; and that, having done this, Mr. Simon, referring to the two periods, 1746-55 and 1846-55, goes on to note that the "so-called 'fever' of the present day occasions only a death-rate of 385 per 100,000, whereas a century ago its death-rate was close on 539." But here Dr. Collins and Mr. Picton have omitted the other figures essential to the comparison. While fevers fell from 539 to 385, small-pox, within the same periods, fell from 204 to

34. Thus, while fever diminished from 100 to 71, small-pox diminished from 100 to 17.

"Sanitation."

Continuing this argument regarding fever, the dissentients point out (Section 21) that since 1871-75 there has been much reduction in the fatality from the diseases registered respectively as typhus, typhoid, and simple fever; and contend that this reduction is probably "due to greater sanitary activity." I do not suppose that any member of the medical profession, no matter how earnestly he may believe that the fall in small-pox is due to vaccination, would doubt that the fall in fevers—in so far as it is not a question of nomenclature—is due to sanitation. "Sanitation" here, however, is a term which has to be interpreted according to circumstances. For typhus fever, sanitation means sufficient food, cleaner houses, and less overcrowding; and for typhoid fever, sanitation means the proper removal of excrement, and the safeguarding of food and drink from the typhoid infection. But it has never been shown that quality and quantity of food, or methods of dealing with excrement, have any particular relation to the spread or prevalence of small-pox. And though it is true that fresh air is of use against both small-pox and typhus, yet the intensity and persistence of the contagion of the former disease, and its carrying power through the atmosphere, are much greater than that of typhus; so that measures relating to individual dwellings, and even to streets and neighbourhoods—improvements in air space and ventilation and cleanliness—which are enough to prevent the spread of typhus, have no equal influence on small-pox. Small-pox indeed is, on the whole, much more comparable with measles and whooping-cough and scarlet fever. In pre-vaccination times small-pox was, like them, a malady of childhood, and its methods of extension are somewhat similar. Yet these diseases have not diminished as small-pox has, in spite of their being open to the same sanitary influences that have lessened typhus and typhoid fever. A Table of measles statistics from 1838 to 1894, given in Section 156 of the Commission's Report, shows that "in the case of measles there has not been during the period in question any diminution in the mortality corresponding with that displayed in the case of small-pox." Regarding scarlet fever the Report says, after giving full statistics: "Neither in England and Wales, nor in Scotland or Ireland, has that death-rate, however, exhibited a decline comparable with the decline in the death-rate from small-pox which has distinguished the period now under consideration, viz.: in

The Zymotics of Childhood.

England and Wales the period from 1838 onward; in Scotland from 1865 onward; and in Ireland from 1841. We find, indeed, that in England and Wales, in Scotland and in Ireland, there was no sign of any decrease in the death-rate from scarlet fever until about 1880, or shortly before that year."

The dissentients argue:

> "Our knowledge of the history, epidemiology, and behaviour of measles and whooping-cough does not suggest to us that they have been very amenable to sanitary reforms, or that they present an analogy with small-pox like the fevers, and especially typhus fever" (Section 20).

It is not obvious here whether the term "sanitary reforms" is intended to include hospital isolation, but that matters little. It is true that measles and whooping-cough have not been found very amenable to "sanitary reforms", but that shows no want of analogy with small-pox; nor does the fact that fevers have been amenable prove their analogy with small-pox. In the passage quoted, Dr. Collins and Mr. Picton simply beg the question.

Section 29 is as follows:—

> "We are led to believe that, but for the disease being kept alive by inoculation, the improvement of the public health which set in towards the end of the eighteenth century, in obedience to the causes to which we have alluded, would have brought about an earlier and greater decline of small-pox mortality. The mere substitution of an non-contagious process like vaccination for the old inoculation, in a population of whom some 80 per cent. or more had acquired, naturally or artificially, such protection as previous small-pox affords, would have a striking effect upon the small-pox death-rate by reducing the liability to infection of the remaining susceptible."

Mr. Picton in the "Contemporary Review."

The description of vaccination as a non-contagious process will be of interest later on, and need not further be referred to here. The statement, however, that when vaccination replaced small-pox inoculation, only 20 per cent. or less of the population stood in need of protection against small-pox, "80 per cent. or more" being already protected by small-pox, is of interest when contrasted with a statement

made in the *Contemporary Review* for October 1896 by Mr. Picton himself. The subject he is discussing in the *Review* is the influence of vaccination in the diminution of small-pox mortality early in the century, and he states that "it is in a high degree unlikely that vaccination reached 50 per cent. of the whole population by 1825"; while with regard to the percentage of the population which *required* protection at this time, he says: "It is absurd to assume every one had small-pox in the last century"; and again, "we are told, indeed, to remember that in the first quarter of the century we must add to the minority vaccinated a considerable number who had recovered from small-pox in the previous century, and were thus protected. This may be so to a certain extent, but *they must have been the minority of the whole survivors from that century;* and, considering the mortality of those days, the number could not have been large in the years 1820-5, from which the vaccination estimates are mainly taken." The italics are mine. No notice seems taken here of those who had had small-pox by inoculation. It would thus appear that, when Mr. Picton is arguing, as in the *Contemporary Review*, that the amount of vaccination early in the century is insufficient to account for the decline in small-pox, he holds that only a minority of those who had survived from the last century had been protected by small-pox. On the other hand, when in the Report of the Commission he desires to argue that the fall in the small-pox mortality early in the century was due to diminution in inoculation, he states in support of his argument that 80 per cent. or more of people living at the end of the eighteenth century had already had small-pox. It is true that in the course of his article in the *Contemporary Review* he admits, with regard to the Statement to which he adhibits his signature for presentation to Parliament, "that all parts of that Statement implying professional knowledge are by Dr. Collins". Whether the statement about "80 per cent. or more" is included among those implying professional knowledge I do not know, but Mr. Picton might at least have tried to recollect, when writing for the *Contemporary Review*, the contents of the document which he had already signed, whether on his own responsibility or trusting to Dr. Collins.

As to the contradictory calculations "80 per cent. or more" and a "minority", it seems to me that all the evidence goes to support the former and not the latter. Small-pox inoculation was certainly very prevalent towards the end of the century, whether or not it was "almost universal", as

Gregory declares. At the same time the number of *deaths* by small-pox in London in the thirty years 1771-1800 was 57,268, so that the recoveries may have amounted to 170,000.* The population "within the Bills" in 1801 was 742,000,† and, deducting those already protected by small-pox (including many who had had it previous to 1771), it would not take a very great amount of inoculation among those still requiring it to protect up to 80 per cent. of the total population. Moreover, inoculation by no means ceased in the early part of the present century, so that the population requiring vaccinal protection was comparatively limited; and even the amount of vaccination quoted by Mr. Picton for places like Cambridge and Norwich would be sufficient to include a large part of the otherwise unprotected population, and to account for a large diminution in the mortality from small-pox. When we come to Gloucester in recent times, however (to continue this digression), the case is very different. Here, until vaccination fell into abeyance, there was little small-pox, and there was no inoculation; and when small-pox struck the town, much of the existing vaccination, even if it amounted to nearly 70 per cent. as Mr. Picton says, was of old date and therefore of diminished value, so that there was ample room for a severe outbreak. Mr. Picton's Gloucester analogy will, indeed, not bear examination, any more than his contradictory statements about the numbers of the protected.

Regarding "the improvement of the public health which set in towards the end of the eighteenth century" (Section 29), there might be endless discussion, as the "allusions" by the dissentients cannot be looked on as evidence. If vaccination had been introduced about 1780 instead of at the end of the century, it would have been equally open to its latter-day opponents to declare that diminution in small-pox following on vaccination was due to sanitary improvements; and a quotation could have been adduced in proof from Dr. John Moore, who in 1786 spoke of "the streets and houses of most of the cities, and of London in particular, being more spacious, dry and airy, from their being infinitely more cleanly in consequence of the new method of paving; from the inhabitants not being so much crowded together; from their being more cleanly in their houses and persons; from

* Taking Mr. Picton's 16 per cent. fatality rate, the recoveries would be over 300,000.

† Simon's *Papers*, Appendix H.

the poor in particular being more commodiously lodged, more salubriously nourished, and better taken care of in all respects." (Q. 5,493.) On the other hand, when we come to the end of the century, to the time of the actual introduction of vaccination, we find a description of London by Dr. Robert Willan, in 1801, quoted in Dr. Creighton's evidence, which tells a very different tale. Willan says that efforts to prevent fever "are often superseded," and that—

Willan on Sanitation, 1801.

"Among the poor the mortality from this cause" (the malignant contagious fever) "was very considerable, notwithstanding the attentive administration of proper articles of diet and of suitable remedies, with plenty of wine. The good effects of all these applications are often superseded by the miserable accommodation of the poor with respect to bedding, and by a total neglect of ventilation in their narrow crowded dwellings. It will scarcely appear credible, though it is precisely true, that persons of the lowest class do not put down clean sheets on their beds three times a year; that, even where no sheets are used they never wash or scour their blankets and coverlets, nor renew them till they are no longer tenable; that curtains, if unfortunately there should be any, are never cleaned, but suffered to continue in the same state till they drop to pieces; lastly, that from three to eight individuals, of different ages, often sleep in the same bed, there being, in general, but one room and one bed for each family. To the above circumstances may be added that the room occupied is either a damp cellar, almost inaccessible to the light, and admitting of no change of air, or a garret with a low roof and small window, the passage to which is close, and kept dark in order to lessen the window-tax, and filled not only with bad air, but with putrid excremental or other abominable effluvia from a vault at the bottom of the staircase. Washing of linen or some other disagreeable business is carried on, while infants are left dozing and children more advanced kept at play whole days on the tainted bed; some unsavoury victuals are from time to time cooked; in many instances idleness, in others the cumbrous furniture or utensils of trade with which the apartments are clogged, prevent the salutary operations of the broom and whitewashing brush, and favour the accumulation of a heterogenous fermenting filth. The rooms do not change their condition until they change their tenants. Often, indeed, so little care is taken that enough of the contagion remains to infect all the inmates who successively occupy the same premises. I recollect a house in Wood's Close, Clerkenwell, wherein the *fomites* of fever were thus preserved for a series of years; at length an accident cleared away the nuisance. A house notorious for dirt and infection, near Clare Market, afforded a further proof of negligence; it was obstinately tenanted till the walls and floor, giving way in the night, crushed the miserable inhabitants. From all these causes combined, there is necessarily produced a complication of fætor, to describe which would be as vain an attempt as for those to conceive who have been always accustomed to neat and comfortable dwellings. The above account is not exaggerated; for the truth of it I appeal to the medical practitioners whose situation or humanity has led them to be acquainted with the wretched inhabitants of some streets in St. Giles's parish; of the courts and alleys adjoining Liquor-pond Street, Hog Island, Turnmill Street, Saffron Hill, Old Street, White-cross Street, Grub Lane, Golden Lane, the two Brick Lanes, Rosemary Lane, Petticoat Lane, Lower East Smithfield, some parts of Upper Westminster, and several streets of Southwark, Rotherhithe, etc. It cannot be wondered at that in such situations contagious diseases should be formed, and attain their highest degree of virulence. The inhabitants of the second storey in houses occupied by the poor are usually better accommodated, and therefore experience, during sickness of any kind, the best effect from public and private charities. But persons thus stationed suffer from

contiguity and from their friendly attentions to those above them, or to the tenants of the cellars; so that in whatever part of the house a fever commences, it is soon diffused among all the inmates and their occasional visitors, especially in seasons which favour its progress, like the last autumn and winter. It is a melancholy consideration that in London and its vicinity hundreds, perhaps thousands, of labourers, heads of families and in the prime of life, are thus consigned to perish annually, being often so situated that medical applications or cordial diet cannot in any way alleviate their distress."

This was written regarding the more easily controlled contagion of fever, and without any reference to small-pox, and it indicates how much assistance sanitation was prepared to give to vaccination in London.

War and Small-pox.

In Section 31, still another cause is suggested by the dissentients to aid in explaining, independently of vaccination, the reduction of small-pox mortality early in the century—

> "The experience of the last century as well as the present indicates a relationship between war and small-pox, and it is not improbable that the fall of small-pox in the earlier part of this century in Europe may have been due in some measure to transition from war to peace . . .".

Seeing that the wars of Napoleon continued up till 1815, and that they included such events as the return from Moscow, and seeing that it was only in 1798 that the method of raising armies by compulsory conscription was first introduced, the selection of "transition from war to peace" as a cause of diminution in small-pox from 1798 to 1825 seems hardly a happy one.

Jenner and the Beginnings of Vaccination.

In Section 33 the dissentients begin the discussion of Jenner and his opinions. There is no good reason to doubt Jenner's statement that the subject had occupied his attention for twenty-five years, nor that he disclosed his hopes and fears to his friend Gardner in 1780. It is known that in 1788 he took up with him to London, and showed to John Hunter and others there, a drawing of a cowpox sore. In 1789, we find from the Minutes of the Gloucestershire Medical Society (*British Medical Journal*, May 23rd, 1896) that he was occupied with his favourite subject of small-pox prevention by inoculative means—the particular means in this case being the insertion of matter derived from a remarkable "sport," as it might now be called, of small-pox, which prevailed in Gloucestershire

at that time. It is evident that in his paper for the Royal Society, as originally written but never formally sent in, he did not attempt to record all the instances he had met with of insusceptibility to small-pox as a result of cow-pox; for when his *Inquiry* was published in 1798, it contained a good many more cases, and most of these had occurred before his earlier paper was prepared. Regarding the four cases of vaccination which Jenner himself tested by means of small-pox inoculation, the dissentients state that it was these "results upon which Jenner based the claim that insusceptibility to small-pox was conferred by inoculation of cow-pox." The statement is in conflict with the facts. These four cases formed only a small part of the grounds for his claim—his paper of the previous year had contained only one such case—and the cases were given only in confirmation of other evidence: evidence, namely, of resistance both to small-pox inoculation and to attack by small-pox among persons who had undergone cow-pox in the natural way, such evidence occurring not only in Jenner's own experience, but in the experience of other practitioners and of the dairy population.

The Variolous Test.

Discussion of the variolous test is begun in Section 39 of the Statement.

Section 40 says that—

> "It is difficult to arrive at any numerical estimate of the proportion of cases of inoculation, in the modified form in which it was practised at the end of last century, in which little or no eruption of pustules appeared upon the body."

This difficulty is not very easy to appreciate. No doubt Sutton himself, the author of the "mild" method of small-pox inoculation, or rather a mercenary clergyman whom he employed to advertise his success, held that absence of eruption in his practice was frequent. But all impartial evidence as to the results of his methods is to the contrary. Sir George Baker writes as if eruption were a matter of course in Sutton's hands. Woodville assumes that because in his own practice of "vaccination" at the Small-Pox Hospital only 300 patients out of 500 had eruptions, therefore vaccination was much milder than small-pox inoculation. Goldson, a critic of vaccination, looked on 100 postules as a mild result of small-pox inoculation, and Ring declared that a country physician had mentioned the absence of eruption as an

extraordinary phenomenon; while Brown, of Musselburgh, an advocate of variolation, looked on eruption as its normal result. Then regarding statistics, there is the Report of the Newcastle Dispensary, giving a tabular statement of variolations from 1780 to 1800, when the mild or Suttonian methods were in vogue, and in a total of 3,181 cases only 6.8 per cent. were without eruption.

Efforts to Attenuate Small-Pox.

In the latter part of Section 40, Dr. Collins (for in the *Contemporary Review* Mr. Picton explains that this part of the Statement is wholly by his colleague) makes a collection of various recorded peculiarities in the behaviour of inoculated small-pox, and sets up these as a standard by which to judge the variolous test. In 1805, Adams, of the Small-Pox Hospital, took up the line of inquiry which Jenner had dropped in 1789, as to the possibility of cultivating a mild inoculative disease from a mild "sport" of small-pox. The "sport" in question was, owing to its appearance, named pearl-pox, by Dr. Adams; and he makes the curious and interesting statement that this peculiar form of disease gave on inoculation a vesicle, presenting the local features not of variolation but of vaccination. In one of Adams's books* there is a drawing of the result, presenting the rounded edge of the vaccine vesicle, without the beaded appearance which characterises variolation by ordinary small-pox lymph. From the registers, this appearance would seem to have been given at once, and not as a result of any attempted attenuation by cultivation from arm to arm, as spoken of by Adams.

Adams.

Guillou.

A Frenchman named Guillou, in 1827, ran short of vaccine lymph in the course of a small-pox epidemic. He seems to have conceived the idea that the body of a vaccinated youth might have the same effect in modifying the small-pox virus that Jenner had all along suspected or believed the cow to possess, and which later experiments, both by Ceely over half a century ago and quite recently by various observers, have actually proved the cow to possess. Guillou, therefore, took matter from the varioloid eruption of a vaccinated girl about 15, and reported that on using this for inoculation vaccine vesicles were produced. On repeating his experiments, however, he found that there was not wanting in some cases an accompaniment of general eruption with fever.

Thiele.

Thiele, of Kasan, possibly thinking that cow's milk might have an effect in altering variola into vaccinia, so as to produce the variolæ vaccinæ found

* *A Popular View of Vaccine Inoculation*, 1807: Appendix.

on the cow's udder and teats, preserved small-pox lymph for ten days between glasses sealed with wax, then diluted it with warm cow's milk, and inoculated it. In the first case he said that the results, both local and general, were considerable, but that after ten removes the pustule had become equal to a vaccine vesicle; and that when the secondary fever ceased to follow such inoculations, the vaccinator could safely proceed to use the matter without further attenuation. With a curious assumption of identity of all these remarkable results, Dr. Collins says that "this mild variety" of small-pox had been observed by Jenner, and had been used under the name of swine-pox. The main point, however, in the present connection is, that these efforts were purposely made in order to obtain a mild small-pox; while in the case of the variolous test, as applied to vaccination all over the world, the purpose was exactly the opposite: namely, to insert small-pox in such a way that if the vaccinated remained capable of suffering from small-pox that disease would unmistakably follow. These minimum and exceptional results, therefore, have no bearing upon the interpretation to be placed upon the variolous test.

Jenner's First Tests.

The suggestion follows, however, by Dr. Collins—though it is hardly expressly stated—that Jenner, in testing his first cases, applied the test in such a way as to yield a minimum result. It will be recollected that (as a preliminary to this suggestion) Dr. Collins has wrongly asserted that it was these four cases, purposely tested, upon which Jenner based his claim that inoculated cow-pox was a preventive of small-pox. The suggestion is equally groundless with the assertion. It is true that in case 3, Jenner took the matter from an early "mother pustule", or rather from an early vesicle, the vesicle of inoculation. But the very reason for his selection of *early* matter was that it should not be in a purulent condition, but pure and limpid, and therefore uncomplicated and efficacious. If Dr. Collins means that the mother pustule was used for the purpose of producing a mild or fallacious result, or that it was likely to produce such a result, he should have given evidence to that effect. In doing so he should have shown that the Greek women, who were the inoculators in Constantinople when Timoni first reported on inoculation, and who got exceedingly mild results, were wrong in thinking that it was of no consequence whether the mother pustule, or matter from the secondary eruption, was used; and that Blake, and Schultz, and Frewen, and Woodville, were wrong

in holding the same view. He should also have shown that Dimsdale, in those abnormally mild results of inoculation which he thought so extraordinary and therefore specially reported on, had used the mother pustule; and he should have explained how, if this was so, Dimsdale himself declares that he "gives a preference to matter taken during the eruptive fever"; and how, in inoculating the Empress of Russia, as to whose safety he was most anxious, he chose matter at the eruptive stage of the disease. Similarly, if Dr. Collins means that the use of clear and limpid lymph instead of pus was likely to give a fallaciously mild result, he should have produced proof; and in doing so he would have had an opportunity of explaining the fact that some inoculators in the last century purposely avoided pus, in order to prevent failure of inoculation. But, instead, Dr. Collins at once goes on to refer to cases whose analogies are entirely opposed to his own line of argument on this point. He says:

The Stonehouse Cow-pox.

> "It is also clear that cow-pox lymph from one of the same stocks used by Jenner (the Stonehouse cow-pox), and in his hands stated to be protective, in the hands of neighbouring surgeons, when submitted to the variolous test, failed to prevent the development of inoculated small-pox in the usual way."

Now the fact is that this Stonehouse "lymph", when used by the other surgeons, was not lymph at all, as we would now understand it, but pus, taken on the twelfth day by one surgeon from the arm of a girl vaccinated by Jenner from the cow; and taken by the other surgeon (Thornton) from the cow itself five days after Jenner had got his own supply, Thornton's source of matter being "a purulent pock which was the only one that was not degenerated into a sordid and painful ulcer." * This was before Jenner had formulated his golden rule for successful vaccination—that in order to secure *its* efficacy, lymph, or clear and limpid fluid instead of pus, should be used. With regard to vaccination, surely even Dr. Collins will agree that Jenner's purpose was that vaccination should be efficient, and to that end he used lymph instead of pus; yet he seems to hold that when Jenner was wanting to test his vaccinations by the variolous test he used lymph instead of pus, in order that the test might fail.

* See p. 187.

Dr. Collins admits that Jenner's Inquiry was favourably received, but goes on to give the names of some medical men who desired more evidence, and of others who were doubtful; and he even cites Moseley, the author of the famous opinion that the face of a vaccinated boy, as to whom he was consulted, was being transformed into the visage of a cow,—the author also of an infamous suggestion as to the likely production and propagation of brutal sympathies and unmentionable proclivities among the vaccinated. In so far, however, as Jenner's views in these early days were subjected to legitimate criticism, the case for vaccination emerged all the stronger.

Having given these references, Dr. Collins immediately proceeds:—

"Thus the matter stood."

> "Thus the matter stood when, in January 1799, cow-pox was discovered in a dairy in the Gray's Inn Lane, London, and attracted the attention of the leading medical men in town, and became the subject of experiments on a large scale by Drs. Woodville and Pearson at the Small-Pox Hospital." (Section 45.)

The statement "Thus the matter stood" is a remarkable one. It absolutely ignores one of the most important and valuable pieces of evidence in the whole history of vaccination. Immediately after Jenner's Inquiry was written, Dr. Pearson, of St. George's Hospital, took up the subject, and wrote to a large number of correspondents in various parts of England, putting to them a series of questions regarding cow-pox in the dairy districts and its alleged anti-variolous powers. In response he received many replies from authoritative and unquestionable sources, more than confirming Jenner's statements regarding the traditions of the dairies. Many medical men told Pearson that, in their own experience and that of others, milkers who had suffered from cow-pox were found insusceptible to inoculation by small-pox; and it at once appeared that not only in Gloucestershire and the neighbourhood, but in the counties of Devon, Dorset, Somerset, Hants, Buckingham, Leicester, Stafford and Oxford, and in London, the belief in the anti-variolous power of cow-pox existed among dairy people, and not unfrequently among medical men practising in dairy districts. Moreover, Dr. Pearson, in presence of Dr. Woodville, the inoculator at the Small-Pox Hospital, himself tested three London milkers who had had cow-pox. These resisted the test, though they "were inoculated in each arm

Pearson's Inquiry.

with perhaps a larger incision and more matter than usual." All this was published in 1798, but in telling how "the matter stood" in January 1799, Dr. Collins is silent regarding Pearson's work. Of less moment is his omission to note the fact that Mr. Cline, the well-known surgeon, had received lymph from Jenner, and with it had vaccinated a patient who afterwards withstood the variolous test repeatedly applied. Unfortunately, Cline had failed to propagate lymph from this source, and cow-pox having temporarily disappeared from Jenner's district, and the series of arm-to-arm operations having been discontinued when he went to London regarding the publication of his Inquiry, Jenner was left without any supply of lymph to satisfy the numerous inquiries which had been evoked by his publication.

A Faulty Sequence.

In Section 46 there is set down by Dr. Collins a sequence of events, which does not accord with the facts. Having stated that Woodville published, in May 1799, the results of his experiments at the Small-Pox Hospital, and that Pearson, in March of the same year, distributed hospital lymph to some 200 practitioners, Dr. Collins writes:—

> "Woodville's cases merit careful attention, as from their number and detail, and from the fact that he had submitted nearly all of them to the variolous test within three months of their 'vaccination', and found they resisted it, they produced a profound impression on the mind of the public and the profession. In July 1800, thirty-three of the most eminent physicians, and forty distinguished surgeons of the metropolis, signed a declaration to the effect that 'those persons who have had the cow-pox are perfectly secure from the future infection of the small-pox, and that the inoculated cow-pox is a much milder and safer disease than the inoculated small-pox.' (*Morning Herald*, July 19th, 1800.)"

The suggestion that the opinion of these seventy-three medical men in July 1800 was based on Woodville's cases reported in May 1799, is without justification. Before July 1800, Woodville's experiments had been criticised and his blunders exposed by Jenner, and Woodville himself had altered both his practice and his teaching. Had cow-pox been an eruptive disease, as Woodville at first foolishly imagined and as Jenner flatly denied, it is not the least likely that the profession would have signed any such

declaration in its favour; and as regards the application of the variolous test, it went on long after Woodville's change of practice. The medical opinion of July 1800 was not indeed a too hasty professional committal on the subject, but was called forth in consequence of criticisms which were being levelled at vaccination by other medical men, no doubt less eminent and distinguished than the seventy-three.

The pedigree of Woodville's hospital matter given by Dr. Collins in Section 50 contains nothing relevant to the question at issue. He is quite unable to show that vaccination on the arm will yield anything else than vaccine lymph, even though immediately previous to or about the time of vaccination, the individual had been infected with small-pox in the ordinary way, or by small-pox inoculation on another part of the body. In Jenner's time, it quickly came to be known that variola and vaccinia locally inserted on different parts of the skin of the same individual would each take its own course, and each produce its own virus; and that matter taken from these two separate sources would, when inserted in other individuals, produce each its separate and unequivocal result.

Woodville's Reports.

Woodville's Reports dealt with 500 cases. Three hundred of these had more or less of a general eruption, and, putting aside those of the 300 in which there were only pimples and papules, the pustules were, as Jenner from the first insisted, pustules of small-pox. Regarding the 500 cases as a whole, the Commission is of opinion that, excepting where the inoculated matter had itself been derived from these pustules of small-pox eruption, it was cow-pox and not small-pox that was inserted; and that the appearance of small-pox eruption on the body had nothing to do with the insertion, but was an incident dependent on infection by the atmosphere of the small-pox hospital in which Woodville's operations were conducted; or, in a few cases, dependent on the application of the variolous test before the cow-pox had run its course. Dr. Collins, on the other hand, thinks that the whole of the 500 cases were due to small-pox inoculation in mistake for cow-pox.

Looking to the eruptions that occurred in Woodville's practice, and to his early statements that these were due to cow-pox, it is clear that his evidence, instead of being "in confirmation of the views advanced by Jenner", as stated by Dr. Collins (Section 47), was in one essential respect the very reverse. Speaking of the lymph issued by Woodville and Pearson, Dr. Collins himself says:—

> "Its effects differed from those of inoculated cow-pox as described by Jenner, and as observed since, in that in the majority of the cases detailed in Woodville's Reports pustules appeared on the body similar to small-pox pustules." (Section 48.)

Even this, however, cannot be accepted as it stands, containing as it does a very obvious *petitio principii.* The main question at issue between Dr. Collins and the Commission with regard to Woodville's cases is, whether the pustules which appeared on the body were or were not the "effects" of the lymph used for inoculation. Dr. Collins is of opinion that they were: the Commission is of opinion that they were not; and the matter is one for evidence, not for assumption.

Dr. Collins points out that there was an eruption in some of the first cases inoculated at the Small-Pox Hospital with lymph understood to be taken from the cows at Gray's Inn Lane, but this tends to confirm the view that the eruption was a result of the hospital atmosphere rather than of error in using small-pox matter instead of cow-pox, or in using an unclean lancet. It is most unlikely that Woodville would have made such a mistake at the very beginning, in a matter so important; though it might be argued that in the course of his subsequent operations he might occasionally have become careless. He himself points out that he began by using a newly-ground lancet. With regard to the allegation that in one case the eruption commenced on the inoculated arm, Dr. Collins adduces no evidence that it was the habit of the general small-pox eruption following on inoculation to commence on the arm, and I am not aware that any such evidence exists in all the literature of inoculation, though, as everyone knows, the mother pustule of inoculation was itself surrounded by satellites. Dr. Collins says:

> "The absence of pustules in two-fifths of the cases does not prove these to have been of other than variolous origin. Woodville's tables show that when he inoculated from pustules on the body (*i.e.*, secondary small-pox pustules, as they are now admitted to have been), he yet in several instances obtained only a local pustule on the arm as the result." (Section 50.)

One might ask in passing why it is that Dr. Collins states that these secondary pustules are "now admitted"

to have been small-pox, when the fact is, that from the very beginning Jenner insisted that they were small-pox, and that in the course of a very short time everyone, even Woodville, came into practical agreement with Jenner on this particular point. No useful purpose can be served in speaking of longstanding assertions as recent admissions. But the dissentient Statement contains no evidence that any large series of inoculations ever resulted in 40 per cent. of non-eruptive cases, as did these operations performed by Woodville. It is quite true, as Dr. Collins says, that in certain of the cases in which Woodville used small-pox matter taken from eruptions on the body, there was no general eruption. But if we pursue this question in order to ascertain whether the proportion of such cases was two-fifths of the whole, what do we find? The facts are that in 66 cases directly inoculated from pustules, 62 had a general eruption and only 4 had no eruption. Not 40 per cent., therefore, but only 6.1 per cent. of these were without eruption, and this 6.1 per cent. is even less than the 6.8 per cent. of non-eruptive cases recorded in the many years' experience of small-pox inoculation at the Newcastle Dispensary (see Table).

Inoculations from Eruptive Pustules.

Cases operated on directly from Pustules of General Eruption.

Source.	No. of Pustules of General Eruption.	No. of Persons operated on with matter therefrom.	Of the Persons thus operated on	
			Had Eruption.	Had no Eruption.
Buckland ...	24	1	1	0
Price	6	4	4	0
Dixon	174	6	6	0
Turner ...	1,000	1	0	1
Streeton ...	300	4	4	0
Platford ...	1,000	6	5	1
H. Timms (1st list)	165	2	2	0
Spooner ...	150	2	2	0
Stringer ...	2	1	1	0
H. Timms (2nd list)	165	9	9	0
Lee	3	16	14	2
Bradley ..	?	14	14	0
		66	62	4

But all these cases, with their high percentage of eruptive cases, are included in the 500, of which 300 had eruptions. Deducting them, the percentage of eruptive cases in the remainder becomes correspondingly reduced. Yet Dr. Collins holds that all alike are variolations. The

view of the Commission, that use of matter derived from the general pustular eruption is the proof of variolation, is strongly supported by the different percentages of eruptive cases among the two classes.

Elsewhere* I have pointed out that the distinction between the local pustule of small-pox and the local vesicle of cow-pox must have become well known to the profession even so early as 1799, and 1800, and subsequently. The very issue of the *Medical and Physical Journal* which contains Pearson's letter to his correspondents enclosing a thread of lymph, gives also three coloured drawings of cow-pox. It shows the cow-pox vesicle in a rounded and regular form, which could not be confused with that of small-pox. Woodville himself learned the difference very early. When he went to Gray's Inn dairy for his lymph, he took with him a copy of Jenner's own plates, to guide him and to show to Sir Joseph Banks, the President of the Royal Society, and the others who accompanied him. In the course of his Reports (his earliest publication on the subject), he writes thus regarding the local appearances of cow-pox and small-pox:

Delineations of Cow-pox Vesicles.

"However, the local tumour excited from the inoculation of the Cow-Pox is commonly of a different appearance from that which is the consequence of inoculation with variolous matter; for if the inoculation be performed by a simple puncture, the consequent tumour, in the proportion of three times out of four or more, assumes a form completely circular, and it continues circumscribed, with its edges elevated and well defined, and its surface flat, through every stage of the disease; while that which is produced from variolous matter either preserves a pustular form or spreads along the skin, and becomes angulated and irregular, or disfigured by numerous vesiculæ."

This is a particularly interesting statement. The mention of the ratio "three times out of four or more", shows that Woodville had not always found what he was regarding as vaccinia to produce the typical vaccine appearance. The natural suggestion is that exceptions might be expected, looking to the fact that towards the end of his 500 cases, on which he is here reporting, he had not infrequently been using matter from the general eruption. But in the first removes from the cow there may well have been some vaccine vesicles which showed their kinship to small-pox by some modification in shape

* *British Medical Journal*, May 23rd, 1896.

or course, and Collingridge's case, in which there were supernumerary vesicles, may be an example of this. He is very clear, however, that the two diseases could proceed *pari passu* on the same subject and yet preserve their distinctive characters. In the same publication he says:

Concurrent Variolation and Vaccination.

"The general character of the tumour formed by the inoculation of the small-pox is very different from that of the cow-pox; and though on the same day a person be inoculated in one arm with the matter of the cow-pox and in the other with that of the small-pox, yet both tumours preserve their respective characteristic appearances throughout the whole course of the disease. This is certainly a strong proof that the two diseases, in respect of their local action, continue separate and distinct."

Once more, in Section 52, we have the assertion by Dr. Collins that Woodville's matter "produced eruptions". This I have already dealt with, and quotations from critics of Woodville, writing twenty years after these events, will not alter what is ascertainable at first hand from Woodville's own Reports. But while Dr. Collins gives evidence that Woodville's experiments continued to be cited as having involved vaccination in the risk of falling into disrepute in 1799, he gives no evidence in support of his own assertion (Section 51) that Woodville's 500 cases "for nearly a century have been cited as demonstrating the truth of Jenner's doctrine."

The Bumpus Lymph.

Jenner himself got a sample of lymph from Woodville (Section 53). It was taken from the arm of a girl, Anne Bumpus, at a date when there was on her no eruption of small-pox from which to take it. She afterwards had 310 pustules, due, as the Commission believe, not to the lymph inserted in her arm, but to concurrent small-pox, caught at the hospital. Dr. Collins states that:

> "In the first case inoculated by Jenner with this matter, pustules appeared on the face; and in the second case, though there was no eruption, the local pustule assumed 'the variolous character', and the areola was studded with minute vesicles."

It seems necessary to explain that the cases thus described as first and second were not two successive removes, but were both directly operated on with the Bumpus lymph. Jenner and his compeers used the word "pustule" very indefinitely. Jenner said he had never seen *maturated pustules*, resulting from either casual cow-pox or inoculated cow-pox, and, in consequence, he wanted to test Woodville's

lymph, as to which he had heard these strange stories of eruptions. He got the lymph, and gives detailed notes of the two cases, paying particular heed to any appearance of eruption. In one, that of Stephen Jenner, on the tenth day three "spots" were visible on the face, and next day "two spots on the face are gone, the other barely perceptible": and again, on the fourteenth day, "two fresh spots appear on the face." Though in the language of the period these "spots" could be described as "pustules", it might have been better if Dr. Collins had not so used the term without explanation in the present day, and on a historical point to which he evidently attaches importance. It is impossible that spots which were "gone" or "barely perceptible" on the second day were ever pustular. Regarding the other case, as to which Dr. Collins writes "though there was no eruption, the local pustule assumed 'the variolous character', and the areola was studded with minute vesicles," Jenner's own notes are, on the tenth day: "The vesicle more elevated than I have been accustomed to see it, and assuming more perfectly the variolous character than is common with the cow-pox at this stage"; while on the eleventh day Dr. Collins's statement becomes correct, that the areola was studded with minute vesicles. These appearances, which are even yet seen occasionally and exceptionally after vaccination, were, as has been already remarked, probably more common in the early removes from the cow, and we never again hear of them in the history of Jenner's lymph. Jenner had always held that while vaccinia and variola were very different, they were related and had resemblances to each other. It happens that the *Medical and Physical Journal* gives contemporaneously a drawing of the results of the Bumpus lymph, taken a day or two later than the supply sent to Jenner; and it would be difficult to describe the results there depicted as being those of small-pox inoculation. They certainly are very different from Kirtland's drawings, reproduced in the *British Medical Journal* (May 23rd, 1896). Regarding the local result in the case of Stephen Jenner, the first of the two cases done directly from this Bumpus lymph, Jenner has this note on the thirteenth day: "The pustule delineated in the second plate in the 'Treatise on the Variolæ Vaccinæ', is a correct representation of that on the child's arm, as it appears at this time." This plate of Jenner's shows the usual circular vesicle of vaccination, as we know it at the end of the century.

As to the statement that there was no other evidence

for vaccination "on a scale at all comparable to that of Woodville", it appears to me that the evidence collected and published in 1806 by Dr. Robert Willan, one of the most distinguished medical men of the time, and one of those who had visited the Gray's Inn dairy along with Woodville and Sir Joseph Banks, is on a scale very much broader and stronger than Woodville's.

(Section 53) Jenner got this Bumpus lymph in February, and the fact that half a year later, he at a particular date chanced to be out of lymph, and subsequently sent some of the Bumpus stock to his correspondent, does not in the least show that he maintained no other stock; but it does show that he had found the Bumpus lymph quite trustworthy, and not productive of eruptions. It is a recorded fact that Marshall used the Kentish Town lymph very largely, as well as the Bumpus lymph; but it chanced that when Jenner got Ring's letter he had neither stock "here in a fit state", as he wrote. Why may he not have had both stocks inserted on the arms of children, among his patients, none of the vesicles being at the proper stage for opening, and no stored lymph in sound condition being at hand?

Jenner's criticism of Pearson in 1812 (Section 54) does not constitute first-hand evidence of what was done in 1799, and Dr. Collins himself would be the first to object to such evidence if presented on the other side. It is true that in the country there were a few cases in which matter sent from the Small-Pox Hospital produced or was followed by variola. Looking to the nature of some of Woodville's proceedings, this is not surprising. But these were exceptional cases, and even as exceptions they were few, and were nearly all comprised within a very short period of time. Some of them, moreover, given in the references mentioned by Dr. Collins, are fully accounted for by the presence of small-pox in the very houses in which the lymph was used, so that these houses had, like the hospital, a small-pox atmosphere (Q. 25,393). The bulk of the evidence regarding country practice is indeed quite the other way.* Within a month after the publication of his Reports, Woodville himself had begun to see his error, and he wrote to the *Medical and Physical Journal* in June 1799, pointing to "a conclusion widely different from that published in the Reports"—the Reports having been published not earlier than the middle of the previous month. In the first series

* See *Brit. Med. Journ.*, vol. i, 1896, p. 1273.

of cases tabulated in the Reports, it is ascertainable that whenever Woodville used eruptive matter he immediately discontinued it, with the exception of a single second remove, and it is only in his second series that he continued it from one remove to another. Both series of cases are comprised within a period of three or four months.

Pearson's Lymph.

Regarding Pearson's lymph, it has been too readily assumed that it was all from the Small-Pox Hospital. He got lymph not only from the Gray's Inn source, but from Willan's dairy in Marylebone Fields. Pearson was the founder of the Vaccine Pock Institution, and in a Paper issued by that Institution in the end of 1811, it is stated: "It is now twelve years since the original matter for the Institution was taken from the cows by Dr. Pearson, and we can perceive no deterioration or difference in its effects." It is true (Section 57) that in issuing his 200 threads of lymph on March 12th, 1799, he states in his accompanying letter that eruptions had been observed in some cases. But the point of interest is, not so much what he wrote to his correspondents, as what they found in using his lymph; and in the October number of the *Journal* of the same year we get his report on the point.

"From my correspondents I have not had a single case of eruption like the variolous since that of Dr. Redfearn's, of Lynn; not one of this sort in Dr. Kelson's, of Sevenoaks, report of about 100 patients; not one in Dr. Mitchell's, of Chatham, of about 50 patients; not one in the report of near 100 patients from Dr. Harrison, of Horncastle, communicated to the Right Hon. Sir Joseph Banks; and, in short, not one case with these eruptions appear in the accounts from my other correspondents."

The Redfearn exception is among those relied on by Dr. Collins. As to the matter sent abroad by Pearson, while Stromeyer, of Hanover, who reported only a comparatively small number of cases, found "small pimples" which, Dr. Collins might have added, "disappear within a day, or two days at furthest," De Carro, of Vienna, one of the principal vaccinators and distributors of lymph on the Continent, found that the results were like those depicted in Jenner's Inquiry. The matter sent by Pearson to Paris died out, and when Woodville (in the year 1800, after he had recognised that he had blundered) carried a new stock thither, it did not produce eruptions.

The Hospital Atmosphere.

That the Commission are right in attributing the eruptions at the hospital mainly to atmospheric infection, is very strongly supported by a statement quoted from

Woodville by Dr. Collins, regarding repeated reversions to the cow as a source of lymph, with the result that "the effects of all the matter I tried were perfectly similar; and pustules proved to be not less frequently the consequence of these trials than with those made of the matter formerly employed." And surely the question is disposed of by what Dr. Woodville says in the paragraph previous to that quoted from by Dr. Collins: "I had not long practised the vaccine inoculation at the Hospital, before I was requested to extend it into private families in the metropolis, where I soon discovered that the cow-pox uniformly appeared in its mildest form, and was never attended with eruptions. I also supplied several medical gentlemen with the vaccine matter, which was used by them with the like result." Thus the lymph which was often followed by eruptions when used in the Small-Pox Hospital, was not followed by eruptions when used outside. The Hospital, therefore, and not the lymph, was the cause of the eruptions.

One of the stocks of lymph used by Woodville at the Hospital, and whose "effects were perfectly similar," was "obtained from Dr. Jenner, and originally taken from Clark's cow." This is the Kentish Town lymph which Jenner used, and some of which he gave to Marshall, who used it largely; and the fact that *at the Hospital* its use in three cases was followed in one of the three by an eruption of "about 100 variolous-like pustules," indicates once more that the Hospital atmosphere was to blame for the eruption.

The World's Vaccine.

The question as to what stocks of lymph were distributed and propagated throughout the world is dealt with by Dr. Collins, and also by the Commission, and their opinions do not agree; but the Commission find it impossible to suppose that Woodville's original stock from Gray's Inn Lane, after passing through the Hospital, was used to the exclusion of other sources. Woodville himself had often reverted to the cow; Pearson had the lymph from Willan's farm; Jenner had the Kentish Town lymph, and later on other lymph, apparently of direct equine origin; and there is no reason to suppose that many unrecorded stocks were not also in use. De Sacco cultivated natural cow-pox on the plains of Lombardy. Besides the lymph distributed by Woodville and Pearson after the exposure of the early blunders, Jenner sent lymph to Vienna, to Berlin, to Professor Waterhouse in America, to Barbadoes, and to Newfoundland; and Ring records that he distributed Jenner's lymph to various places on the Continent and

America (Commission's Report, Section 27). Prof. Crookshank shows in his evidence (Q. 11,298) that cow-pox was a well-known disease in the beginning of the century in many foreign countries — Italy, Holland, Denmark, Germany, North America, etc. The subject of vaccination was being eagerly discussed, and it is impossible to doubt that many stocks of lymph were used and tested and propagated all over the world.

The Woodville and Pearson episode has been dealt with above at inordinate length, relative to its real importance, but those who wish to follow it further can do so in the very able discussion in Appendix B of the Commission's Report. My reason for going into the question in such detail is, that within the last few years the operations of Woodville and Pearson have been forced into unnatural prominence by opponents of vaccination, and it seemed desirable to traverse what Dr. Collins has to say regarding them. It has, in effect, been argued by anti-vaccinationists (*a*) that Woodville's lymph, supposed to be derived from the Gray's Inn Lane dairy, was entirely variolous; (*b*) that this lymph constituted the world's supply of so-called vaccine; (*c*) that it was the application of the variolous test to Woodville's cases which convinced the profession of the value of vaccination; and that, as Woodville's cases were really variolous, the test was fallacious, being performed not on vaccinated but on variolated persons. I have endeavoured to show that (*a*) and (*b*) are incorrect, and it is necessary now to turn to (*c*). In Section 60 the dissentients say:—

> "Apart from the vague statements of Marshall, which must be received with reserve, we are unable to find in the early days of vaccination any large body of definite evidence sufficient to establish the contention of Jenner that cow-pox, and especially that of equine origin, affords, when conveyed to man, security from the future infection of small-pox."

What evidence the dissentients would consider sufficient to establish Jenner's contention I do not know, but it may be useful to indicate the evidence they look on as insufficient.

In the first place, the abundant evidence contained in *Pearson's Inquiry*, published in 1798, is entirely passed over. Apart from equine origin, that evidence gave very strong support to Jenner's contention that cow-pox, con-

veyed to man, afforded security from future small-pox.

The Testing of Variolation.

It is necessary to note what were the proofs of failure or success in times when small-pox inoculation was itself occasionally tested by repetition of the operation. Here there is evidence that the result of variolation of a person protected against small-pox might vary within well-recognised limits. There might be no local effect whatever; there might be early inflammation running a rapid course and rapidly terminating; there might be a pimple or a small vesicle or pustule, and the matter in this vesicle or pustule might itself be capable of giving small-pox by inoculation, the person on whose arm the matter had been locally cultivated being himself all the while immune to small-pox infection, and not affected either to feverishness or to eruption. Fever and general eruption, or in a small number of cases fever without general eruption, but with the local result of inoculation, were the evidences that the person had not been immune, and that his previous inoculation had failed.* It is true that in the course of a very extensive practice, Dimsdale had found a few persons who were so refractory to primary inoculation when performed as a protection against small-pox, that only a little local inflammation and pustulation, with more or less disturbance of the system, resulted; and there was a percentage—apparently six or seven per cent.—of people whose reaction to primary or protective inoculation consisted only in the local pustule accompanied by the primary fever of small-pox. But the local pustule of small-pox inoculation, as depicted in Kirtland's plates and by Pearson and other authors, was a very different thing from the small pimple or pustule that could be produced on the arm of a person previously rendered immune to small-pox. The local pustule of a successful small-pox inoculation ran a regular course of a fortnight or thereby, and had beaded edges and a surrounding zone of satellites. The local pustule on the arm of a person already protected was small in size, and ran a shortened and abortive course. All such facts were known and recognised by the profession when its knowledge of variolation was equal to our knowledge of vaccination; and there is no reason for any confusion on the subject through citation of the peculiar cases

* Blake's *Letter to a Surgeon*, 1771; Schultz's *Account of Inoculation*, 1767, pp. 122, 124; Mudge's *Dissertation on the Inoculated Small-pox*, 1777; Ring's *Treatise*, p. 359; *Report of the Surgeons of the Edinburgh Pock Institution*, 1809.

of absence of ordinary reaction to primary inoculation which puzzled Dimsdale.

The Profession and the Test.

Coming to the Variolous Test as practised by the profession after Woodville's errors had become matter of professional knowledge, we find that Woodville himself continued to apply it. In 1802 he stated to a Committee of the House of Commons, that there had been vaccinated up till 1st June of that year in the hospital "7,500; about one half of which was since inoculated with small-pox, *in none of whom did the small-pox produce any effect.*" The italics are his own. It was in the latter part of the year 1800 that he had written to the *Medical and Physical Journal,* stating that he had repeatedly reverted to the cow, and used different stocks of such matter in the hospital. To the Committee he also declared his belief that general eruptions never did follow vaccination, so that his tests had been applied to non-eruptive cases. In an early publication (*Observations*, 1800) Woodville makes an interesting statement. After his 500 cases which we have already so fully discussed, he reported that he had operated on 1,000 more, and had tested them "with the like result," *i.e.,* the same result as he had got in testing 400 of his first 500. Now, many of these first cases were protected by their variola, inoculated or concurrent. But the 1,000 subsequent cases, done when he had come to see that he had made mistakes, gave "the like result." This means, if I read the story aright, that the vaccinations when tested gave the same results as the variolations when tested: so that the two processes had, at least for the time, produced the like immunity against inoculated small-pox.

Willan's Testimony.

Willan, in 1806, makes this general statement regarding the experience of private practitioners, in vaccination and in testing vaccination. (*Vaccine Inoculation,* p. 13).

"If the numbers vaccinated by private practitioners could be laid before the public, and their success ascertained, we should, I am convinced, obtain a most satisfactory proof of the advantages of vaccination. On making an enquiry with this view of the medical gentlemen whom I have had the pleasure to meet in the course of the last three months, I have learned that those mentioned below [a list of 86 medical men] have vaccinated more than 18,000 persons who have since remained free from the small-pox, though most of them were afterwards either inoculated with variolous matter, or exposed at different periods to contagion."

Willan himself saw 180 tests performed, and he gives many elaborate details and publishes a plate showing the local results produced, which in no case make the slightest approach to the local results of an ordinary small-pox inoculation in an unprotected person. Hill, of Portsea, published (1801) details of ten cases tested at various intervals after vaccination, and the resulting local inflammation was so early and short-lived as to prove insusceptibility. Tierney (afterwards Sir Matthew Tierney) wrote to Jenner (*Continuation of Facts and Observations*, p. 25), regarding his practice in the Navy, that he had tested four cases, and he describes results similar to Hill's. Later on, Lord Berkeley wrote to Jenner, giving the result of testing two of Jenner's own vaccinations. There was early inflammation, which died away without producing any effect (*Med. Phys. Jour.*, vi, p. 105). The Rev. Mr. Holt (*Med. Phys. Jour.* Vol. ii, p. 303) tested six cases and got the early inflammation, disappearing without the formation of a pustule. His lymph came from Woodville, subsequent to his letter to the *Journal* correcting the false conclusions derivable from his Reports. The Rev. W. Finch tested twenty cases (*Med. Phys. Jour.*, iii, p. 419) and "all entirely resisted the infection." His lymph was from Woodville, in the summer following his letter to the *Journal*. Kelson got matter from Pearson in the autumn of 1799 (his 200 threads had been sent out in March), and tested forty cases with the above-quoted result of early local inflammation, dying away again. Barry, of Cork, got matter from the Editor of the *Journal* (iv, p. 248) in April 1800—a year after Woodville's 500 cases. He vaccinated 250, of whom 247 had no eruption, and he tested twelve with variolous matter "from a child covered with variolous eruptions in a state of suppuration". Again the result was early inflammation, without even a local pustule. Woodville took lymph to Paris in July 1800, and the French reporters said "this disease excites no general eruption" (*Med. Phys. Jour.*, iv., p. 255, and v. p. 101). Nineteen cases were tested "with fresh pus" from "a variolous infant," and "deep incisions" were made, and more variolous matter was introduced into the incisions at various times, with the usual absence of result, or trifling local result: the most being a local pustule capable of conveying small-pox by inoculation, as could sometimes be done from matter locally cultivated on the skin of persons previously protected by small-pox.

Woodville's Lymph Tested.

Pearson's Lymph Tested.

Pearson had sent one of his 200 threads to Liverpool,

and it was used on April 3rd, 1799, but failed. No more was done there until February 1800, when lymph was obtained from Jenner, and formed the commencement of the practice, which was quickly taken up and extended. Up to the year 1806, Willan (p. 31) records that Dr. Park, of Liverpool, had vaccinated 499, and had tested twenty or thirty early in his practice, but they all resisted the disease. Mr. Dale had vaccinated 663, and tested 150 of them by variolation, "without producing the small-pox in a single instance". Many others had tested a few cases with a similar result. Jenner's Lymph Tested.

Passing over several years' evidence, and coming to periods as to which it is sometimes alleged that the inoculation test had ceased to be practised, the surgeons of the Edinburgh Pock Institution reported in 1809 that they had lately tested children vaccinated eight or nine years before, and found "that they completely resist the disease". At the Foundling Hospital (*Med. Phys. Jour.*, xvii, p. 488), twenty-one children who had been vaccinated on April 10th, 1801, were tested on 9th August, 1802, also on 13th November, 1804, and February 23rd, 1807, "without any consequence except slight inflammation of the inoculated part in a few instances, and in three cases a small pustule on the part where the matter was inserted". Later Tests.

Dr. John Rollo, Surgeon-General to the Army, published in 1804 a very elaborate report of a series of tests of small-pox and cow-pox, his object being to ascertain whether the vaccinal protection was permanent, and he declares— Rollo's Tests.

"That the vaccine inoculation resists the action of the variolous disease; that when this had been apparently diminished as a preventative power, the variolous disease has been much milder and less complete, but how far time may further influence the vaccine power, time alone can decide; though at present it remains entirely in favour of vaccination, as deducible from our Report".

Ring, in his *Treatise* (p. 997), makes the interesting statement that Sutton, the famous inoculator (who would then be a very old man), whom he had introduced to Jenner, vaccinated twenty cases, and tested them, but "had not been able to produce the small-pox on those who had undergone vaccination". In his *History of the Norwich Epidemic* (1820), Cross states that "several hundreds" vaccinated at various periods from the beginning of the century, were tested by the variolous test during the epidemic and resisted it, excepting that a few pimples appeared in about one case in forty or fifty. Sutton's Tests Cross's Tests.

Equine Lymph Tested.

As regards the testing of the protective power of equine lymph by subsequent variolation, if only equine matter was used in India, as Dr. Collins argues (Section 59), then the following statement will apply (Shoolbred's *Report on Vaccine Inoculation in Bengal*, London, 1803, p. 24-5)—

"Accordingly on the 16th of January, 1803, in the presence of Mr. Munro, second Member of the Medical Board, I inoculated with recent fluid variolous matter taken on the spot three children who had previously passed through the vaccine disease, and three more with the same matter only an hour after it had been taken. These inoculations, as was expected, produced nothing like variolous affection. In some there occurred a slight inflammation of three or four days; in others no visible effect followed insertion of the matter .... Similar proofs of the efficiency of vaccination in preventing small-pox both by inoculation and exposure were obtained by other gentlemen in other parts of the country."

Letters giving the results of tests similarly resulting are referred to Dr. Shoolbred, but are not quoted in the printed Report.

There is in the medical journals of the early part of the century much more evidence of the use of the variolous test, but enough has been quoted above to indicate how the matter stands. Lymph issued by Jenner and Woodville and Pearson and others was subjected everywhere throughout the world to the variolous test, and the conclusion was that cow-pox protected against small-pox.

A Dilemma.

At this point in their Statement, the dissentients find themselves in a dilemma. We have seen that they urge very strongly that a main reason for the decline of small-pox in the early part of the present century was the diminution in the practice of small-pox inoculation. But when they come to the introduction of vaccination, they adopt the view that so-called vaccination was really variolation; that Woodville's and Pearson's early lymph was variolous lymph; and that that lymph "was the great source from which in the years 1799 and 1800, and perhaps later, the practice of 'vaccination' was started" (Section 52), and "would appear to have been the chief source of the first stocks of lymph used on the Continent and in America" (Section 58). The next proposition in this thesis would naturally be that the decrease in variolation in the early part of the century was not real but only apparent, and that to what remained of admitted variolation there had to be added the new practice of

so-called vaccination; and that moreover, as "vaccination" was universally looked on as non-infectious, even the imperfect precautions which had been taken with regard to acknowledged variolation were neglected with regard to variolation under its new name. Thus, instead of small-pox being checked, it ought to have been more widely spread, and have followed "vaccination" wherever it went. But small-pox did diminish, and the dissentients have argued that it diminished owing largely to diminished inoculation of small-pox, so it is obviously impossible to follow the path that lies plainly before them if they are to avoid the pitfall to which it leads. Here is the byeway by which they seek to get out of the difficulty:—

> 61. "Whatever may have been the nature of the matter used and widely distributed by Woodville and Pearson, and even if we must regard it all as derived from small-pox, it seems that after a time, whether from attenuation or dilution of the original matter, or the selection of mild cases, or from other causes, the operation gradually ceased to be followed by pustular eruptions, was no longer infectious, and came to assume the local phenomena now observed in ordinary vaccination." And in Section 63: "We therefore, in using the term 'vaccination,' must be held to employ it colloquially, and not exclusively as an equivalent for cow-poxing."

Having thus looked the difficulty more or less boldly in the face, and thanking God that they are well rid of such pestilent questions, the two pilgrims prepare to pass on. By-and-by they find occasion to retrace their steps, and, having forgotten the danger, we shall see (p. 144) that they tumble right in. Meanwhile it seems necessary to detain them for a moment. In Section 52 we were told that the hospital matter of Woodville and Pearson "was . . . . variolous." Now (Section 61) we are suddenly reduced to the hypothetical position, "even if we must regard it all as derived from small-pox." We have seen how rigorous have been the demands of Dr. Collins and Mr. Picton for absolute proof regarding the origin of stocks of lymph, and the nature of the cases submitted to the variolous test. Now, when the point has been reached at which it is necessary to depart from the view that alleged inoculated cow-pox was really small-pox, and to adopt the view that a non-infectious disease was being propagated

instead of an infectious one, and that a non-protective operation had been substituted for a protective one, and that vaccinia with all its alleged dangers was beginning to be carried throughout the world, there is no longer, to the dissentients, any need either for proof or probability. "It seems that, after a time, whether from attenuation or dilution of the original matter, or the selection of mild cases, or from other causes", and so forth. Any time or no time in particular; any one of several speculations or no speculation in particular, is now quite enough for those who heretofore have refused to be satisfied by anything short of mathematical demonstration. In short, looking to the line of argument adopted up till this point, what justification is there for the dissentients determining that, onward from the lymph distributions by Woodville and Pearson in the very beginning of the century, they shall use the term vaccination "colloquially"? In particular, why do they say they shall now use it "colloquially, and not *exclusively* as an equivalent of cow-poxing"; the fact being that up till now they have held it to mean not cow-poxing but small-poxing? To follow their argument, however, we must pass on without getting in the Statement any satisfactory reply to these questions.

Bryce's Test. In Section 64 there is a passing reference to Bryce's test, which Jenner recommended in 1808. This test was the exact analogue of that which had occasionally been used in the previous century for small-pox inoculation: the test, namely, of making a second insertion of lymph after the first insertion had been three or four days on its course, and observing whether the local vesicle had a shorter cycle of development, so as to overtake the original vesicle and reach maturity at the same time. Its principle was that early and shortened inflammation and vesiculation showed that the system was under the influence of the previous protective operation. Jenner's recommendation was sound and logical. Even previous to 1808, at Pearson's institution (the Vaccine Pock Institution)—

"Many trials of this kind were instituted publicly . . . in 1807, in the presence of several Fellows of the College, to show that the re-inoculation with vaccine matter was equally a test of security of the constitution against the small-pox as variolous matter."*

The next few Sections need not detain us: they are

* Sanders, *A Comprehensive View of the Small-Pox, Cow-Pox, and Chicken-Pox*, Edinburgh, 1813, p. 107. Also *Medical Physical Journal*, December 1811.

devoted to proving part of the modern doctrine of vaccination, namely, that the operation does not necessarily give life-long protection. The doctrine, of course, should have its fruits in practice—the practice of re-vaccination—but the dissentients do not carry it that length. The need for re-vaccination, however, is illustrated by the reference to Copenhagen (Section 70) where, after small-pox had been entirely absent from the time when primary vaccination became the practice, until 1823 or thereby, it began again to show itself among the vaccinated.

Section 71 is as follows :—

Small-Pox Diminution after 1800.

> "The fall in small-pox death-rate observed in many places in the first vicennium of this century can hardly be ascribed to vaccination. If the limited and voluntary practice of the operation could be so influential upon small-pox mortality as such a theory demands, it is strange indeed that the more complete and compulsory adoption of it should have been so uninfluential against recurring epidemics as was especially exemplified in the pandemic of 1870-74, and against more recent outbreaks in this country and abroad, in which the vaccinated figured largely among the victims."

For a reply, all that is necessary is to turn back to Section 29, where, London being under consideration, they speak of—

> "A population of whom some 80 per cent. or more had acquired, naturally or artificially, such protection as previous small-pox affords."

In such a population "the limited and voluntary practice" of vaccination would be very influential; while in 1870-74, with infantile vaccination as the mainstay, a large section of the population would have outgrown their original protection, especially against attack, though as to fatality rates even the adults were much benefited. For the same reason, the want of re-vaccination, it is true that many vaccinated patients are now admitted to hospital (Section 72); and that even severe and fatal small-pox may occur among the vaccinated (Section 73). It was known to occur in the last century among persons who had had small-pox (Section 75). It is true that the Select Committee of 1871 reported, as quoted by the dissentients (Section 75)—

Protection by Small-Pox.

> "That the almost universal opinion of medical science and authority is in accordance with Dr. Gull, when he states that vaccination is as protective against small-pox as small-pox itself."

But it is also true, as not quoted by the dissentients, that both the Select Committee and Dr. Gull approve of re-vaccination, Dr. Gull saying "Certainly, I do recommend it" (Q. 4760), and that this must be kept in view in interpreting the quoted statement.

This should be borne in mind also in valuing the opinions quoted in Section 76, where the dissentients say—

> "We cannot recall a single witness who has been examined by us on this question who has not admitted that, whatever may be the amount of protection afforded by vaccination, it is at any rate less than that conferred by a previous attack of small-pox."

They might also have added that they could not recall a single witness to whom they had put the question, using the term vaccination as including re-vaccination. They quote Mr. Sweeting "as of opinion that vaccination is decidedly less protective," but what Mr. Sweeting replied was: "Decidedly less; infantile vaccination is less protective to adults than previous small-pox." Even this did not not draw from Dr. Collins the query regarding vaccination as inclusive of re-vaccination.

The Attack Rate amongst the Vaccinated and Unvaccinated.

The discussion of this question begins in Section 78. With reference to admissions to small-pox hospitals the dissentients say (Section 79)—

> "And in proportion as the ratio of the vaccinated to the unvaccinated in the hospital approximates to that obtaining outside (assuming the admissions to be a fair sample of the whole cases) we must regard the protection against attack of small-pox as approximating to *nil*."

The Highgate Hospital.

The heart and soul of this statement is contained in the parenthesis. The example selected is the London Small-Pox Hospital, and we would presume it to be distinguished by its compliance with the condition laid down, that "the admissions" should be "a fair sample" of the

facts in London outside the Hospital. A table is given, in which it is shown that the percentage of the vaccinated among the admissions rose steadily from 1826 to 1888-91, the admissions in these last three or four years amounting to only fourteen, all of them vaccinated. For comparison another table is given, showing that from 1872 to 1881 the practice of vaccination increased in London, the default diminishing from 8.8 to 5.7, while from 1881 to 1892 the practice steadily diminished, the default increasing year by year from 5.7 to 18.4. Between 1885 and 1888-91, the hospital figures, such as they are, show that the percentage of vaccinated admissions had increased from 93 to 100; while in London the practice of vaccination had diminished so considerably that the default, which was only 5.7 per cent. in 1881, mounted to 16.4 per cent. in 1891. If this proves anything, it surely proves too much. *Primâ facie*, one would expect that vaccination being a protection, but not an absolute protection, the proportion of small-pox attacks among the vaccinated in a community would increase with increase of the vaccinated population in that community, and would decrease with decrease. Here, however, we find vaccination in London diminishing since 1881, while vaccinated admissions to the London Small-Pox Hospital are increasing. A statement of one or two facts may furnish an explanation of the anomaly. The admissions were to be a "fair sample" of the population outside, but the only evidence furnished on this essential point is to be found in the relation of the two names, London Small-Pox Hospital and London. It is a fact well known to everyone who knows the hospital arrangements for small-pox in London, that the London Small-Pox Hospital is *not* now the hospital for small-pox in London. It has had no pretensions to be so for the past quarter of a century. The word "Small-Pox" has been included in its title since its foundation in 1746, but now it is a name and little more. All but a mere fraction of London hospital-treated small-pox has been so treated, not in the London Small-Pox Hospital, but in the various hospitals belonging to the Metropolitan Asylums Board. Yet some small-pox—and until a few years ago a good deal of small-pox—*has* been treated in this hospital even since 1871, and the next question is: Does this furnish a "fair sample" of London small-pox outside the hospital? In two essentials it does not. The first is, that only paying patients are admitted, sent in by a class of the population containing more than the average percentage

of London vaccination, and therefore not furnishing in its hospital admissions a fair sample as required by the dissentients. In any special class of population vaccinated up to 100 per cent., it is obvious that when small-pox occurs, all the cases it sends to hospital will be vaccinated cases. The second point is even more important. We have seen that in the opinion of the Commission "the highest protective potency" of vaccination to prevent attack by small-pox might be fairly stated to cover a period of nine or ten years, after which, they say, it "rapidly diminishes." This, of course, does not refer to its more prolonged potency against death by small-pox. It is obvious that, accepting this view, the fairest sample would consist only of admissions under ten years of age, and the basis of comparison would be the percentage of the vaccinated in the London population of the same age, from which the hospital cases were drawn. Obviously, also, the most unfair sample that could be found would be the admissions to a hospital which excluded the cases occurring in the early years of life, and admitted only those whose period of highest protective potency had more or less nearly expired. But this is exactly the sample hit upon by the dissentients. No cases are admitted to the London Small-Pox Hospital under seven years of age. I assume that Dr. Collins and Mr. Picton could not have been aware of this radical flaw in their argument when they prepared their Statement, and that Mr. Picton would not subsequently have repeated the argument in full in the *Contemporary Review* had he even then known how the matter really stands. They have indeed unwittingly furnished an excellently fair sample of the exception which proves the rule. I have been quite unable to ascertain how long this regulation has existed at the Highgate Hospital (as the London Small-Pox Hospital is now usually designated, even by the dissentients in their questioning of witnesses, though not in their Statement of Dissent). The regulation is said, however, to go back for "many years," but it cannot have been in force in Marson's time—or at least in the earlier part of it—as he gives statistics of cases under the age of seven years.

The conclusion of the dissentients regarding the Highgate Hospital is:—

> "We are unable, therefore, to infer from the statistics of the London Small-Pox Hospital that vaccination has any very marked effect in reducing the

> liability to attack by small-pox, seeing that the proportion of vaccinated cases to the total has increased with the increasing proportion of the vaccinated in the population."

They do not here allege that they can infer from the statistics anything against vaccination. The statement is merely negative. One would suppose, therefore, that they next would turn to the experience of the other small-pox hospitals of London, which might yield them a definite inference of some kind, especially as they had heard plenty of evidence on the subject. They do nothing of the sort, however, but pass straight away from the exact statistics of hospital admissions to questions relating to epidemics occurring in towns. They pay no heed to such facts as that placed before them by Dr. Gayton, that in Homerton Hospital, of 2,169 admissions under ten years of age, the unvaccinated contributed 1,187, or 47.6 per cent.; nor to the corresponding fact as given by Mr. Sweeting, that of 370 children under ten years admitted to Fulham Hospital, 168, or 45.4 per cent., were unvaccinated. The dissentients do not suggest that the percentage of unvaccinated children in the population from which these hospitals drew their patients approached 47 or 45 per cent. They simply pass by the evidence in silence.

Homerton and Fulham Hospitals.

It is true, as stated in Section 84, that the figures derived from the special Reports relating to infected towns are given in detail in the Report of the Commission, and that in the dissentients' statement, therefore, "it is needless to recapitulate them". But whether it was equally needless to answer them is another matter, and the comments of the dissentients can not be looked on as an answer. The special Reports relate to matters of fact, very fully stated by the reporters appointed by the Commission, including the dissentients.

With regard to Sheffield (Section 85), the allowance that ought to be made in the census of the vaccinated and unvaccinated population for persons vaccinated between the beginning of the epidemic and the date of the census must be matter of opinion: the Commission thinking that Dr. Barry's deductions are amply sufficient, while the two dissentients think otherwise. Seeing, however, that they insist so strongly here on exactitude of statistics, they should not have ignored the statistics of vaccinated and unvaccinated admissions to the wards of Homerton and Fulham hospitals, while they themselves immediately

Statistical Methods.

proceed to vague speculations as to the proportions of vaccinated and unvaccinated in the infected towns.

> "86. Another method of arriving at the proportion of the vaccinated to the unvaccinated in the population would be by reference to the vaccination registers. It is, however, only since 1872 that these have been compiled in their present form."
>
> "In Sheffield, we learn fom Table XCVII (p. 185 of Dr. Barry's Report), that for the years 1878-87, 84 per cent. of the children born during those years were successfully vaccinated, some 10 per cent. died unvaccinated, and 4.5 per cent, remained unaccounted for. But in arriving at a conclusion as to the proportion of the whole population vaccinated on the basis of the vaccination registers, it is necessary to bear in mind that the proportion of the vaccinated amongst those born before the Vaccination Acts of 1853, 1867, and 1871 was in all probability very much less; thus in 1862, at an inspection of the borough school children by an inspector of the Privy Council, 13 or 14 per cent. were found unvaccinated. It would be hazardous to assert, in view of these facts, that the proportion of the vaccinated in the whole population of Sheffield at or about the beginning of the epidemic much exceeded 90 per cent. Now, of the cases of small-pox investigated by Dr. Barry in the epidemic of 1887-8, 4,151 out of 4,703, or 88 per cent., were vaccinated."

The Conclusion Dissented from.

The figures previous to 1872 being unknown, it is easy to make assumptions regarding them; but this would be of little consequence if only the dissentients would, in this part of their Statement of Dissent from the views of their colleagues, give some indication or recognition of what these views really are. Let us turn to Section 377 of the Report of the Commission. Here the conclusion is clearly stated—

"3. That the protection it affords against attacks of the disease is greatest during the years immediately succeeding the operation of vaccination. It is impossible to fix with precision the length of this period of highest protection. Though not in all cases the same, if a period is to be fixed, it might, we think, fairly be said to cover in general a period of nine or ten years.

"4. That after the lapse of the period of highest protective potency, the efficacy of vaccination to protect

against attack rapidly diminishes; but that it is still considerable in the next quinquennium, and possibly never altogether ceases."

This does not refer to protection against death, which is held by the Commission to outlast protection against attack. These conclusions refer to attack only; but it is exactly with liability to attack that the dissentients are here dealing. The question is, whether the Commission are right or wrong in believing that protection against attack lasts in almost full amount for, say, nine or ten years, and in lessened amount for at least other five years. Now, the Sheffield outbreak occurred in 1887. Obviously, therefore, as regards high protective potency against attack, the only relevant statistics are those beginning with 1872, or fifteen years previously; and it happens to be just since 1872 that the vaccination registers are available. Surely in these circumstances the issue is perfectly clear, and the question for the dissentients to solve is (confining the facts to the period of highest protective potency), why did 4.5 per cent. of unvaccinated children under ten years' old in Sheffield contribute 39.2 per cent. of the total small-pox attacks that occurred at that age.* With regard to this opinion of the Commission as to the period of highest protective potency, there is no relevancy in the line of argument followed by the dissentients in which they speculate as to how many children had been vaccinated belonging to previous generations.

Concerning Warrington, Dewsbury and Leicester, the same method is pursued; but it may be worth noting the sequence in which the two statements of the dissentients appear for each town (*a*) their own suppositions regarding the percentage of the vaccinated; and (*b*) the percentages of small-pox attacks contributed by the vaccinated, and ascertained by the Commission's reporters. We have seen that in Sheffield, according to the dissentients, "it would be hazardous to assert that the proportion of the vaccinated much exceeded 90 per cent." This is the calculation, if we may so term it. Then comes the ascertained fact that, "of the cases of small-pox . . . . 88 per cent. were vaccinated". So in Warrington the dissentients "would doubt" whether the vaccinated population "greatly exceeded 90 per cent.": and then comes the fact—that the vaccinated cases were 89.7 per cent. of the whole. Then for Leicester: "If we assume 50 or 60 per cent. of the population . . . to have been

* There were 581 attacks under 10 years, and 228 of these were among the unvaccinated. The 4.5 per cent. is got from the Vaccination Registers.

vaccinated, . . . it is interesting to observe that . . . 55.7 per cent. were vaccinated"; and for Dewsbury: "it would be scarcely safe to assume that . . . more than . . . between 60 and 70 per cent. were vaccinated", "while of the cases of small-pox 64 per cent. were vaccinated." The accuracy of these various calculations is nothing short of marvellous. The 90 per cent. and 89.7 per cent. is a specially close approximation. When one realises, however, that the two calculators were all the while aware of the ascertained facts to which they were leading up, the marvel is lessened. Thus, as they say, "if we assume 50 or 60 per cent.", then "it is interesting to observe" that the ascertained attacks *are* between 50 and 60 per cent. The assumption is indeed interesting—even entertaining.

Section 87 refers to Dr. Coupland's Leicester Report, which is not yet published.

Section 91 is as follows:—

Gloucester Attack Rates.

> "We have not received as yet the report of Dr. Coupland from Gloucester, but from the figures contained in Section 243 of the report of our colleagues, it would appear that from a census made of persons in infected houses 30 per cent. of the vaccinated were attacked, and 46 per cent. of the unvaccinated, nearly all of whom were children under ten years of age."

Even this hardly seems to fall in with the view that vaccination makes no difference between the vaccinated and unvaccinated; but still less is the view supported by Section 244 of their colleagues' report, which gives the statistics for vaccinated and unvaccinated children, thus:—

> "In the vaccinated class there were 272 children under 10 years of age, of whom 24, or 8.8 per cent., were attacked.
>
> "In the unvaccinated class there were 1,331 children under 10, of whom 617, or 46.3 per cent., were attacked.
>
> "In the vaccinated class there were 3,114 persons over 10 years of age, of whom 1,004, or 32.2 per cent., were attacked.
>
> "In the unvaccinated class there were 144 persons over 10, of whom 72, or 50 per cent., were attacked."

In Gloucester, with its great neglect of infantile vaccination for some years previous to the epidemic, these are just the results that the theory of vaccination would lead

us to expect—a fivefold rate of attack among unvaccinated children as compared with vaccinated; and among adults a greatly lessened contrast, owing to the considerable loss of protection against attack through lapse of time amongst the vaccinated.

Section 92 is preceded by the statement that the Gloucester Report had not been received. It is well known that small-pox varies greatly in intensity in different epidemics, and the Gloucester figures being wanting from the table given in the Section, the mild type of disease which struck Leicester is not contrasted with the severe form which seized on Gloucester. The dissentients say "the fact that only 22 or 25 per cent. of the deaths occurred in children under ten is but small compensation to Sheffield and Warrington for their high attack rate and mortality rate." But it is not a question of compensation. It is a question of the interpretation of evidence, and here is the Table (Section 246, Commission's Report) against which this criticism is levelled by the dissentients:

—	Attack Rate under 10.		Attack Rate over 10.	
	Vaccinated.	Un-vaccinated.	Vaccinated.	Un-vaccinated.
Sheffield ...	7.9	67.6	28.3	53.6
Warrington	4.4	54.5	29.9	57.6
Dewsbury .	10.2	50.8	27.7	53.4
Leicester ...	2.5	35.3	22.2	47.6
Gloucester .	8.8	46.3	32.2	50.0

Figures like these speak for themselves, but they are not to be found in the Statement of Dissent.

In concluding what I have to say regarding these sections dealing with the attack rates of the vaccinated and unvaccinated, let me again point out how the matter stands as between the Commission and the dissentients. The proposition from which Dr. Collins and his colleague dissent is that vaccination can be reasonably relied on to protect against attack up to a given age, say fifteen years. In Section 114 there is a statement of attacks of small-pox among vaccinated children in Sheffield, Dewsbury, Warrington, London, etc., but they give no comparative attack-rates of the vaccinated and unvaccinated belonging to this period of life. In so far as their statistics show anything, what they do show is, not so much the failure of

vaccination to protect the early years of life, as the need for re-vaccination to protect against attack after the period of greatest protective potency by primary vaccination has fairly elapsed.

The Fatality Rate Among the Vaccinated and Unvaccinated.

This subject is discussed in Sections 93—110. The first question is as to the fatality of small-pox in the last century. The dissentients state it as varying from one in three to about one in forty. Unless a good deal of historical evidence is to be set aside, one in three is far from the highest fatality rate. In an epidemic in Iceland in 1707, it is recorded that there were 18,000 deaths in a population of 50,000; and if the mortality was 36 per cent., the fatality rate (deaths among persons attacked), must have been still greater; while for Mexico and Greenland, and the aborigines of America, much higher rates are stated. Regarding Jurin's statistics, the dissentients write in Section 94:—

Jurin's Fatality Rate

> "Jurin's figures, based upon a large number of cases collected by him during the first half of last century, give a fatality from natural small-pox of 16.5 per cent. of those attacked. We agree with our colleagues that the criticism made upon Jurin's figures, to the effect that deaths under two years of age were excluded, fails to establish the alleged fact."

Criticism, however, may fail to establish a given thesis, and yet may throw such doubt on the subject-matter criticised as to leave it valueless; and while the dissentients speak of the exclusion of deaths under two years old as an "alleged fact," the Commission (Section 227) speaks of Jurin's fatality rate of 16.5 as itself a "fact or alleged fact." It happens that it was the writer of this paper who criticised Jurin's statistics, and I cannot pretend to judge my own evidence; but I do not see how (in view of Jurin's own statements about children under one and two years old, and in view of the age-period 0-2 years being left blank in the only group of Jurin's statistics—those for Aynho—in which the ages are ascertainable) it is possible even for the dissentients to place any reliance on Jurin's fatality rates.

Dr. Collins and his colleague make no reference to the

obvious and unquestioned fact that the natural fatality rate of small-pox depends on the ages of those attacked by the disease. It is exceedingly fatal in infancy; the fatality rapidly decreases to its minimum in the third quinquennium of life, and then gradually rises again. In the absence of vaccination, therefore, the fatality depends on the age-incidence of the disease. At the same time, again in the absence of vaccination, the age-incidence of the disease could not otherwise than depend on the interval between epidemics. After an absence of twenty or thirty years, when small-pox revisited a community, the ages of the attacked would have a totally different range from that belonging to an epidemic after only a five years' interval. Now, for Jurin's statistics the places are known, but excepting in one instance the ages are not known, and excepting in another instance, the interval since the previous epidemic is not known. In the single outbreak where the ages are known—though the total cases only number 132—it is at once seen that the fatality rates correspond very closely with the rates at the same ages among the unvaccinated in the present day; while in the single instance (Boston, America) where the interval is known (and where the cases were very numerous) the interval accounts for the low fatality rate exhibited. How, then, with the exception of these two places, can any weight be attached to Jurin's fatality rates, especially when it is found that in his different epidemics the fatality ranged from 10 per cent, to 36 per cent.? Just as the type of scarlet fever varies as between one part of a century and another, so the type of small-pox varies immensely at different times and in different places (as recently in Leicester and Gloucester). This being so, any average rate of fatality for a century should be calculated on epidemics extending over many years. With regard to Jurin's figures, one would suppose from the Statement of the dissentients that this condition at least had been fulfilled, seeing that they say the statistics were "collected by him during the first half of last century." But the statement is quite misleading. The figures were collected, not during the half century, but during the seven years 1722-28, and this is the only period to which they refer. Most of these remarks apply also to the dissentients' reference to Bernouilli. The data on which his calculations are based are not given, and it does not appear that any account was taken of age-incidence, or of intervals between epidemics. The comparisons instituted (Sections

97-99) between past and present fatality rates without relation to vaccination are therefore of little or no value.

Fatality-rates of Vaccinated and Unvaccinated.

The fatality rates of the vaccinated and unvaccinated are next referred to.

In Section 100 it is pointed out that in Berlin and Duisburg the rates differed less than elsewhere between the two classes, in the epidemic of 1871-2. The reason is not far to seek. Dr. Seaton (*Handbook of Vaccination*) states that the vaccination law of Prussia had no penalty attached excepting in the event of subsequent small-pox. A frequent result in an epidemic would be that people whose unvaccinated children were attacked by small-pox would state that they had been vaccinated, and so evade the law.

Section 101 contains a statement of the usual anti-vaccination objections to evidence that the fatality rates are higher in the unvaccinated than in the vaccinated.

Anti-Vaccination Objections.

"Thus, this class, it is urged, includes (1) infants under the age of vaccination; (2) those whose vaccination is postponed on account of poor health; and (3) those of the lowest and most neglected social class. Inasmuch as these, when they constitute a considerable proportion of the whole unvaccinated class, would raise the case mortality from reasons apart from vaccination, it is urged that the high fatality of the unvaccinated must not be ascribed merely to the fact of their non-vaccination.

"Objection has also been made to the classification by marks on the skin of cases of a disease whose chief symptom is, and the chief cause of whose fatality is, the abundance of an eruption on the skin. It is claimed that the fact of vaccination or non-vaccination of small-pox patients should be determined by reference to the vaccination registers, not by the visibility of marks on the arm.

"Reference has been made to some of these objections in our colleagues' report, but we hardly think sufficient weight has been attached to them. It is clear that if these objections are well founded, some part of the difference between the fatality of the vaccinated and the unvaccinated is explicable without reference to vaccination. It is difficult

to say whether the whole difference can be thus explained."

The dissentients need have no difficulty in weighing and measuring the value of these objections. The first can be got over, as the Commission does get over it (Sections 217 and 222), by the simple expedient of omitting deaths occurring under one year of age, or by classifying the vaccinated and unvaccinated fatality rates at different ages; and so exhibiting the fact that small-pox in persons above the vaccination age behaves very differently as between the vaccinated and the unvaccinated. The second and third objections are, thanks to anti-vaccination itself, equally easy to get rid of. In places where the vaccination laws have been a dead letter, and where there is an annual default of say 50 to 85 per cent. in the vaccination of infants, it is obvious that the unvaccinated must be fairly typical of the average health and social condition of the community. Indeed, their health should be even better, according to theories which attribute much illness and weakness to vaccination. Neither can the remaining objection throw any doubt on the value of the classification into vaccinated and unvaccinated, for there is a superabundance of statistics in which a middle class of "doubtful" cases receives all regarding whom there need be any question. The example selected (Section 102) by the dissentients to illustrate the first difficulty is Warrington, but, unfortunately, the Warrington Report is not yet published nor open to public comment.

Section 103 urges the objection that children whose vaccination is certified for postponement, owing to ill health, add to the fatality rate of the unvaccinated as compared with the vaccinated. In illustration of this objection the dissentients suddenly alter the whole basis of comparison, namely, the rates among vaccinated and unvaccinated. Badly-vaccinated Leicester having had a mild type of small-pox, and the Report regarding badly-vaccinated Gloucester, with its severe type of small-pox not being in their hands,* the dissentients compare the 12.0 per cent. fatality of mild small-pox among the unvaccinated in Leicester, with the 35.3 per cent. of unvaccinated fatality in Warrington, and the 49.6 per cent. in Sheffield, as to which there is no longer any complaint of errors in the census of vaccination. This comparison of mild and

* The statistics, however, are given in the *Commission's Report*, Section 210.

severe forms of the disease they make in apparent oblivion of the fact that in Section 96 they hold that in times when there was no vaccination, the type varied so much that the fatality rate ranged roughly from 2.5 per cent. to 33 per cent. Obviously, the comparison must be between the vaccinated and the unvaccinated when subjected to the same epidemic type of small-pox. In Leicester the fatality rate of the vaccinated was one per cent., and of the unvaccinated 12 per cent. In Warrington the corresponding rates were 6.4 and 35.3; in Sheffield, 4.8 and 49.6; in Dewsbury, 2.6 and 25.1; in London 2.2 and 24.2; and in Gloucester, 9.9 and 40.9 per cent. Here there are various degrees of severity of epidemic type, but in each epidemic the unvaccinated have a much higher fatality rate than the vaccinated. Here also, in these three centres of anti-vaccination, Leicester, Dewsbury and Gloucester, we have a reply to the contentions detailed in Sections 103 and 104, that postponements under medical certificate, and poverty, may account for the higher fatality rates of the unvaccinated. In presence of such facts it is useless to dispute regarding possible minute influences of poverty, etc., in towns where the unvaccinated may really belong more largely than the vaccinated to the poorer classes.

The remaining objection has regard to doubtful cases. As to confluent small-pox, it is true, as stated by Dr. Birdwood, that in the worst instances the eruption obscures the scars, and that Dr. Savill met with cases in the stage in which this had occurred; but the experience of hospital physicians like Dr. Gayton and Mr. Sweeting is that when cases are admitted to hospital, the eruption has very seldom reached this stage. Here again, not only in nearly all hospitals, but in the three centres of anti-vaccination, the doubtful cases are tabulated as such; and it really matters nothing to the teaching of the figures above given, whether they be kept apart, or added to the vaccinated, or added to the unvaccinated.

Fatality Statistics.

Before leaving the question of fatality, it may be worth while just to indicate the nature and amount of the evidence against which the criticisms of the dissentients are directed. In the six towns specially dealt with, 2,321 unvaccinated persons were attacked, and of these 822 died, or 35.4 per cent. Of cases under 10 years old there were 1,449, with 36 per cent. of deaths; and over 10 years old there were 870, with 34.3 per cent. of deaths. The total attacks among vaccinated and unvaccinated were 11,065, with 1,283 deaths, or 11.5 per cent.; and, deducting the un-

vaccinated, the attacks among the vaccinated were 8,744, with 461 deaths, or 5.2 per cent.

In children under 10, the total attacks both of vaccinated and unvaccinated were 2,338, and the deaths 539, or 26.4 per cent.; of these 589 were vaccinated, with 16 deaths, or 2.7 per cent. In order to meet the objection regarding unvaccinated deaths in the first months of life, let all cases under one year old be omitted, and there are left 570 vaccinated attacks, with 16 deaths or 2.8 per cent.; and 1,235 unvaccinated attacks, with 375 deaths or 30.3 per cent. This calculation, moreover, includes all doubtful cases as vaccinated.

In hospitals, Marson found that in 19,467 cases, the unvaccinated fatality was 36.5 per cent., while the rate among those having only one vaccination mark was 12.8 per cent. In 10,403 cases treated by Dr. Gayton at Homerton Hospital, the deaths among the vaccinated (including all alleged vaccinations, without mark) were 869 in 8,234, or 10.5 per cent., and among the unvaccinated, 938 in 2,169, or 43.4 per cent. At Fulham Hospital Mr. Sweeting had 2,584 cases with a fatality rate of 16·5 per cent., and among the vaccinated (including alleged vaccination), the deaths were 263 in 2,226, or 11.4 per cent., while among the unvaccinated the deaths were 165 in 358, or 46 per cent. The comments of the dissentients are altogether inadequate to explain away such facts as these.

The Severity of Type of Small-pox amongst Vaccinated and Unvaccinated.

This is the next question as to which Dr. Collins and Mr. Picton state their Grounds for Dissent from the conclusions of the Commission.

This is their statement :—

> "109. Attempts have been made to classify cases of small-pox according to their severity as well as according to their fatality. This classification is open to the obvious objection that 'no two men could, independently, classify the same series of cases in the same way.' When a further division of the severe and mild cases into vaccinated and unvaccinated is made, another source of error is introduced by reason of the inconclusiveness of the evidence as to vaccination."

Classification of Type of Small-Pox.

Comment has already been made on the latter of the two contentions here contained. Whether or not the other contention—that no two men could classify the same cases in the same way—has any relevance to the subject depends obviously on the question whether, in the evidence relied on by the Commission, in any given group of cases, the classification of some cases was made by one man, and of other cases by another man. If that were so, the next question would be whether the dissimilarity in methods of classification was so great as to make the classification unreliable for the required purpose. But we never get the length of this question. The above quotation contains all that the dissentients have to say on a subject which occupies eighteen Sections of the Commission's Report. On turning to these Sections (252-269) there is nothing to show that the criticism of the dissentients has any validity. The first group of cases dealt with consists of those occurring in the Winter Street Hospital, Sheffield. The type of disease is stated under four headings, progressive in severity —varioloid, discrete, coherent, and confluent, and the following Table contains the facts as ascertained.

In Sheffield.

Type of Disease.	Vaccinated		Unvaccinated	
	Cases.	Per Cent.	Cases.	Per Cent.
Varioloid	293	35.5	0	0.0
Discrete	413	50.0	50	17.9
Coherent	107	13.0	175	62.5
Confluent	12	1.5	55	19.6

Here, of course, the criticisms regarding the Sheffield census do not apply, and neither Dr. Collins nor Mr. Picton asked a question as to whether the classification had been the work of one or several observers.

The statistics in the following Table may also be quoted (Section 266, *Royal Commission's Report*) :—

		Milder.	Severer.
Sheffield	... Vaccinated ...	82.8 ...	17.2
„	... Unvaccinated ...	18.5 ...	81.5
Dewsbury	... Vaccinated ...	82.0 ...	18.0
„	... Unvaccinated ...	23.1 ...	76.9
Leicester	... Vaccinated ...	81.4 ...	18.6
„	... Unvaccinated ...	27.2 ...	72.8
Warrington	... Vaccinated ...	78.2 ...	21.8
„	... Unvaccinated ...	25.4 ...	70.6

It is needless to comment on the hopeless insufficiency of the reasons for dissent to meet the case for vaccination as

set forth in the statistics contained in the Report; but it may be noted that as Dewsbury and Leicester are hotbeds of anti-vaccination, with the vaccination laws in abeyance, there can be no question of the unvaccinated being poverty-stricken, or constituting a weakly class whose vaccination had been postponed under medical certificate.

THE DRIFT* TOWARDS RENEWAL OF SUSCEPTIBILITY TO SMALL-POX AMONG THE VACCINATED.

While half a dozen lines suffice to contain all that the dissentients care to urge against the views of the Commission on vaccinal protection against severity of type of small-pox, they devote fifteen Sections (111-125 inclusive) to the next question. Here again the reader is impressed with the fact that the difference between the dissentients and the Commission is one of degree, especially when Dr. Collins and Mr. Picton (Section 113) say that—

> "A relatively low fatality rate in vaccinated children under ten is, as is shown in the Report, a remarkable feature in recent epidemics; and this, if it were constant, might well be urged as a ground for encouraging the practice of infant vaccination when small-pox is prevalent, if no other means for controlling the disease were available."

Section 112, which reverts to the variolous test, is as follows:— Vaccination or Variolation?

> "Goldson, of Portsea, in 1804 published cases of the inoculated and the natural disease occurring within two or three years of *vaccination.* In 1809 Brown, of Musselburgh, published his *Inquiry into the Anti-variolous Power of Vaccination;* in which he recorded forty-eight cases of children who had caught small-pox within three, four, five, six, seven, eight, nine and ten years of their *vaccination.*"

Here we find a most remarkable admission. I have italicised the word *vaccination,* which is of special interest. The cases with which Goldson deals were reported by him so early as 1804, and the interval between the original operation and the testing of it was, according to the dis-

* For the word "Drift" I am indebted to Dr. J. B. Russell's "Study of 972 Cases of Small-Pox", in the *Glasgow Medical Journal,* 1872.

sentients, "within two or three years." In certain cases it was over three years, as some of the original operations were done before the end of the year 1800. We have seen how strenuously the dissentients have argued that perhaps all, or nearly all, so-called vaccinations at this period were really variolations, and that the testing of such cases was utterly fallacious *qua* vaccination. Now, however, we come to cases in which it is alleged that the variolous test showed that the original operation had been non-protective; and what follows in the Statement of the dissentients? Notwithstanding their previous contentions, they straightway speak of the original operations as *vaccinations*, not variolations, and so give away the case which they had so laboriously set up. It can hardly be supposed that Dr. Collins and his colleague claim the liberty to use the word vaccination "colloquially" (Section 63), when it happens that the operations, though performed in the first years of the practice, can be presented as examples of failure to protect against subsequent variolation.*

Goldson and Brown.

The dissentients are indeed perfectly justified in speaking of the cases of Goldson and Brown as vaccinations, and they would have been equally justified in using the same name for the thousands of operations which resisted the variolous tests. But when they quote Goldson's cases as failures it is necessary to turn to the relevant facts. At the time in question, it was the habit to vaccinate only in one place, and the vesicle was often small. This, it is now held, yields a protection less enduring than that conferred by three or four vesicles, or a larger area. Moreover, Goldson's stock of matter, as will be seen on consulting his work, had its origin in an adult case from which it was taken on the eleventh day. In some of the cases related by him and Brown the appearances after the variolous test are very trifling—a few pimples, or a local pustule without eruption. And while Brown's work of 1809 is quoted from, no mention is made of the considerable modification which his opinions against vaccination underwent as the result of increased experience, as shown in his Letter to Dr. Gregory, published in 1842.

In Section 114 a statement is given of vaccinated children under ten years attacked by small-pox in Sheffield, Dewsbury, Warrington and London, but nothing is said as to the quality of their vaccination, as evidenced by the area or number or character of the scars, or of the amount

* See p. 125.

of the total vaccinated population among which the cases occurred.

With regard to susceptibility of a vaccinated person to re-vaccination, and the interpretation to be put on such susceptibility, Dr. Collins and Mr. Picton write in a curiously hypothetical fashion in Sections 117—125. Bryce's Test.

> "If we accept, with Jenner and Bryce, the theory that re-vaccination is a test of susceptibility to small-pox 'of equal efficacy' with variolous inoculation, we then have a means whereby we may gauge the duration of the temporary protection or antagonism conferred by vaccination." (Section 117.)

And in Section 122 there is a long quotation from Professor F. Smith, without any statement that the dissentients agree with his views. Jenner and Bryce were not discussing the same subject as the dissentients. Bryce's test was performed merely to see whether vaccination had really "taken." Within three or four days of vaccinating in one place, he would vaccinate in another place; and if the second vaccination ran a rapid course so as to overtake the first, then he held that the first vaccination was successful, the rapid course of the second vaccination being the evidence of success. But this was a totally different thing from holding that the duration of resistance to re-vaccination *after* the original vaccination had run its course, was to be looked on as a measure of the duration of resistance to small-pox by infection in the ordinary way. We have already seen that even in persons whose protection was due not to vaccination but to variolation, it was often possible to produce on the arm a local result, sometimes even a small pustule containing variolous matter capable of conveying the disease to others. This local susceptibility, however, was no evidence that the person was liable to infection by small-pox through the atmosphere. In the same way it is mere assumption to assert that susceptibility to the local phenomena of re-vaccination is equivalent to susceptibility to attack of small-pox by atmospheric infection. And, indeed, the dissentients hardly make the assumption. They leave it rather as a suggestion to the reader, without giving him any hint that the two things may be essentially different. It is true that Layet found that in vaccinated children six years old, about 40 per cent. were susceptible to re-vaccination. But in Sheffield, at even a later average age (5—10 years), when vaccinated children were exposed to infection by living in houses in Susceptibility to Re-Vaccination.

which cases of small-pox existed, not 40 per cent., nor 20 per cent., but less than 10 per cent. were infected ; and the reality of the exposure in these houses is shown by the fact that, among the unvaccinated children of the same age similarly exposed, no less than 92 per cent were seized by the disease. Similarly, a large proportion of children of school age, and of adults,—soldiers (some of whom are pitted with small-pox), sailors, hospital nurses, etc.,—can be re-vaccinated, but this is by no means equivalent to saying that before their re-vaccination they are all equally susceptible to small-pox by infection. But, again, it has to be noted that the dissentients avoid saying so. They begin with an "if," and never get beyond it.

The Influence of Amount and Quality of Vaccination.

This question is dealt with by the dissentients in Sections 126—130 of their Statement, and by the Commission in Sections 272—298 of their Report. There are two main points for consideration (*a*) number and area of successful vaccinal insertions, and (*b*) foveation of the resulting scars. The value to be attached to both or either must be decided, not by conflicting theories as to the nature of zymosis, but by experience. Neither can it be decided according to degree of susceptibility to re-vaccination, as we have just seen that susceptibility to local results of variolation or vaccination is not to be interpreted as showing susceptibility to small-pox by ordinary infection. Marson's well-known statistics (Section 28), showing a striking diminution in the fatality rates, according as the vaccinated had one, two, three or four marks of vaccination, tell in the same direction whether deaths from "superadded disease" be included or excluded; and the statistics of other hospitals support Marson's conclusions. Similarly as to "doubtful" cases, where they have been separately classified, their inclusion among the vaccinated really does not affect the lessons derivable from the tabulations.

Classification of Vaccine Scars.

As with regard to classification of types of small-pox, so with regard to classification of vaccine scars, the dissentients (Section 129) attach weight to the fact that all observers—Drs. Gayton and Sweeting for example—do not follow the same rules. This quite accounts for differences in amounts of "good vaccination" recorded in two different hospitals, one being by

one observer, and the other wholly by another observer. It does not, however, detract from the value of comparisons made amongst facts noted *by the same observer*, in the same institution, or the same outbreak, as it is obvious that the standard of each individual observer can be subject to no appreciable deviation. It is also obvious that different observers may be trusted for comparison or summation where the data consist, not of more or less indefinite terms like "good vaccination" and "indifferent vaccination," but of numbers of marks actually counted on the arm, and of deaths distributed among the classes subdivided on this method. Where small numbers of cases and deaths are dealt with, and are much subdivided, the value of the facts is much diminished, and it would be surprising if percentages of fatality came out always the same for twenties or forties, as in calculations based on thousands of cases. As vaccinal protection diminishes with advancing years, it is not surprising that the comparative influence of number, and area, and character of scars, should be less apparent in adults than in children.

The comments of the dissentients to which these remarks apply are quite insufficient to explain the facts cited by the Commission. From Marson onwards, observers who have had open to them any large field of experience, have found one after another, that the amount of vaccination does very much affect the subsequent protection against small-pox. In the Sheffield Borough Hospital (and here again the census question does not come in), 95 cases with one vaccination mark or with none visible but yet with alleged vaccination, had 13 deaths, or 13.7 per cent.; 259 cases with two marks had 24 deaths, or 9.3 per cent.; 372 with three marks had 21 deaths, or 5.7 per cent.; 99 with four marks or more had two deaths, or 2 per cent. The severity of type of the disease varied similarly. In the Dewsbury epidemic, Dr. Coupland found that there were with one or two marks, 209 cases with 10 deaths; and with three or four marks, 252 cases with one death. Dr. Luff reported that in the London outbreak, among cases with one scar there were 7.4 mild cases to one severe case; but with four scars there were 37.2 mild cases to one severe case; while those with two and three scars came midway with 11.9 and 12.3 mild cases to each severe case.

Classification by Number.

Excluding Marson's cases, and taking only the results of more recent hospital experience, in 6,839 cases the percentage fatality of one-marked cases was 6.2; of two-

marked cases, 5.8; of three-marked cases, 3.7; of four-marked cases, 2.2; and the numbers of cases on which these percentages are based were 1,357, 1,971, 1,997 and 1,514 respectively.

Classification by Area.

As to area of scars, Dr. Luff found that where it was under a quarter of an inch the severe attacks were 12.6 per cent.; where it was between a quarter and half an inch, the corresponding percentage was 4.6; and where it was over half an inch, 3.9.

Classification by Foveation.

As to foveation of scars, Dr. Luff found that where the scars were not foveated, the severe cases were 10.4 of the whole, and where they were foveated, the severe cases were 4.6 of the whole.

The Altered Age-Incidence of Small-Pox.

In Sections 131-148 a strenuous effort is made to discount, as a proof of the influence of vaccination on small-pox, the remarkable change which has taken place in the age-incidence of the disease since pre-vaccination times. Formerly children took small-pox because they were unprotected, and adults were comparatively free from small-pox because so many of them were protected by previous small-pox. Nowadays, it is the children who resist small-pox, and the adults who show liability to be attacked by it; and those who believe in vaccination look on the protection of children as due to vaccination, and the want of protection of adults as due to want of re-vaccination.

It seems likely that more time and toil have been spent in forging the weapons with which to assail this position than on any other part of the dissentients' Statement. In the preparation an enormous mass of statistical material must have been examined, with rejection of everything but the minutest fraction of it, the retained fraction being all that could be found to fit into any thesis that the altered age-incidence of small-pox is to be accounted for by agencies other than vaccination. The onlooker can only marvel at the greatness of the zeal and patience which have been misapplied in endeavouring to prove the worse to be the better cause.

In Section 131 it is stated:—

> "Statistics collected last century, and especially during the inoculation period, when small-pox was almost endemic, seem to show that a large proportion of all children suffered from it, and the deaths from

the disease were mostly those of children. Records of the seventeenth century suggest that the disease at that time was less prevalent, and affected adults as well as children."

But the earliest existing statistics are those for Geneva, beginning in 1580, long before the inoculation period, and going on to 1760. In 25,349 small-pox deaths in Geneva, there were in the first year of life, 6,792; in the second year, 5,416; in the third, 4,116; in the fourth, 2,826; in the fifth, 1,928; and so forth. Over twenty years of age there were, of the 25,349 deaths, only 152; and over thirty-five years of age there was not one. No figures more striking than these belong to the inoculation period. They, however, are not referred to by the dissentients. Geneva Statistics.

Figures are given for the village of Aynho, in Northamptonshire, for the two years 1723-4, in which, of 132 cases (not deaths) recorded, 104 were over ten years old. The purpose here is to show that in rural districts the incidence of small-pox may have been less on children than in towns. It was the writer of this paper who brought these figures before the Commission with the very object of pointing out that the period 0-2 years is left blank in the Table; and the suggestion is that those years are left blank in accordance with Jurin's remarkable views about deaths under two years being set down under such headings as "Chrysoms," "Infants," "Convulsions," etc., without mention of small-pox. It is a truism that exceptions are found to every rule, but it is equally truistic that human affairs are best guided by rules. Alike in villages or small towns, like Pudsey and Kilmarnock, in Chester, Warrington, Manchester, etc., in this country, and in the Hague and in Sweden, small-pox was a disease of childhood, but wherever and whenever there were long intervals between epidemics, the disease would not be confined to childhood. Aynho.

Section 131 continues:—

"It has been pointed out 'that the whole question of the age-incidence of fatal small-pox depends on the frequency of the epidemics. If an epidemic comes once in twenty years you will not have the same proportion of deaths under five years as you have in a place where it comes in a period of less than five years. It all depends upon that; and there is no possibility of getting any general law from isolated places.'" Epidemic Intervals.

The quotation also is from the writer's evidence. It refers to small-pox in pre-vaccination times and also in pre-inoculation times, when there was no protection of the infantile population. Small-pox, like measles, will attack the susceptible at any age, and the age of the attacked obviously depends on the intervals between epidemics. But in Geneva and Kilmarnock and elsewhere small-pox, like measles, came very frequently, so that the children were the sufferers. So it is still with measles; but nowadays when small-pox strikes a community, no matter what the interval may be, the vaccinated children largely escape, while unvaccinated are attacked in a measure altogether out of proportion to their relative numbers.

The dissentients make what looks like a slip in Section 135, where they say—

> "In Sections 171-192 the change of age-incidence has been fully treated in special relation to changes in the law and in regard to vaccination; in this relation it is therefore unnecessary to labour the the point further."

But it is the Commission in their own Report, and not dissentients in their Statement, who discuss the question in Sections 171-192. How then are the dissentients relieved from "labouring the point," unless, indeed, they do not dissent? An example of the statistical material selected with regard to this subject of age-incidence, is found in a Table in which, in order to prove that small-pox mortality, calculated on the population, has increased in adult life, the non-epidemic period 1847-53 is contrasted with the period 1872-80, containing the second worst year of the great epidemic of 1870-72, the severest epidemic that has occurred in all the years since vaccination was made obligatory in this country. Obviously there is more small-pox during an epidemic than at other times, and no Table was needed to illustrate that fact.

In Section 138, regarding London, we have a Table presented thus, with Dr. Thorne's name given in the marginal reference:—

Dr. Thorne's Table.

"*Annual Small-pox Death-Rates per* 100,000 *at different Ages in London.*

	0-5 Years.	5 Years and upwards.
1851-60	130	13
1861-70	116	14
1871-80	113	34
1881-88	37	16"

But on turning to Dr. Thorne's evidence, the impression produced is different. Here is the Table as put in by him:—

TABLE F.

LONDON.

TABLE *showing, since Compulsory Vaccination and during its complete enforcement, the general decline in Small-pox Mortality among the Infant Population, and the fluctuations of Small-pox Mortality among the Population above Infancy.*

Five-Year Periods (for most part).	Annual Small-pox Death-rates per 100,000 at each Age.	
	0-5.	5+
1843–46	213	15
1847–49	244	16
1850–54	150	12
1855–59	101	11¾
1860–64	115	15
1865–69	118	13½
1870–74	192	46
1875–79	62½	24
1880–84	45	19
1885–88	10	5

The fuller and more detailed figures of Dr. Thorne give more information, and yield a more useful lesson, than the condensed reproduction of part of them by the dissentients. It is necessary, however, to observe further how even the condensed Table is used. Here are the dissentients' remarks concerning it.—

> "Thus we see that, except in the last period (which has been one of increasing default in regard to vaccination), and then *only in the case of those under five years of age,* there had been no substantial reduction of small-pox mortality, while *at all ages over five* the mortality from small-pox has been actually greater in the last three periods than in the first. Such saving of life as there has been in London in the period 1851-88 was most noticeable in the period 1881-88, and was confined to children under five years of age."

I have italicised the words to which I wish to call attention. The Table gives no justification for the deductions drawn from it regarding "all ages over five." It is true that by selecting a statement which, for a different purpose,

separates out only the 0-5 years period from the whole of the rest of life, and by avoiding any further analysis of all the life periods above five years, the figures of the second column are obtained. But they show nothing whatever regarding the age periods 5-10 years, 10-15 years, 15-25 years, and so forth. For anything in the Table to the contrary, small-pox might be rapidly increasing, say between 5 and 10 years, and rapidly diminishing, say between 50 and 60 years; and on the other hand exactly the reverse might be the case. Yet the dissentients say "thus we see" that "at *all* ages over five the mortality has been"—and so forth, Dr. Thorne separated out only the 0-5 years period, but he drew no such conclusions regarding "*all* ages" over five. Another Table, very well known, prepared by the Registrar General, submitted to the Commission and printed in their minutes, was equally available to the dissentients, showing various life periods over five, and having the advantage of referring, not merely to London, but to the whole of England and Wales. That Table (p. 114, First Report), would have shown that in the three periods 1847-53, 1854-71, and 1872-87, not only had small-pox death-rates per million living in the first quinquennium of life fallen from 1,617 to 817, and then to 242, but in the second quinquennium of life there had been a fall represented by the figures (1), 337; (2), 243; and (3), 120; and in the third quinquennium a further fall represented by the figures (1), 94; (2), 88; and (3), 69. As to all the other age periods specified, namely, 15-25 years, 25-45 years, and 45 years upwards, while there had been a considerable rise as between the years 1847-53 and 1854-71, there had been a fall as between 1854-71 and 1872-87. And if the dissentients had any objection to the Registrar General's subdivisions of the years 1847-87, Dr. Ogle could no doubt have supplied the figures year by year.*

While, however, they do not seem to care for the Registrar General's facts, the dissentients set great store by his opinion expresed in the year 1879: "That the sanitary efforts made of late years should have more distinctly affected the mortality of the young, . . ." and they accordingly set up the thesis that the change of small-pox age-incidence, from earlier to later parts of life may be due to sanitation. The thesis is ingenious, and an ingenious endeavour is made to support it by analogy. It appears (Section 142), that

Sanitation and Young Lives.

* See Appendix to Mr. Noel Humphrey's *Paper* read before the Statistical Society, Feb. 16th, 1897.

between two quinquennia there was in typhus fever, a diminution in the children's share from 6.4 per cent. to 3.4 per cent. How long it took to effect this diminution does not clearly appear, for though the later quinquennium is 1886-90, the earlier is described only as "the earliest quinquennium which the Registrar General's figures enable us to use." The difference between 6.4 per cent. and 3.4 per cent. seems very trifling, but taking 6.4 as a *centum*, the dissentients state the reduction, not as 3 per cent., but as 46.9 per cent. In enteric fever the difference is greater, and is similarly dealt with. On the other hand, in the *Contemporary Review*, Mr. Picton states that, the attack-rate of the unvaccinated (at all ages) in Gloucester being 46.7 per cent., and of the vaccinated 30.3 per cent.—"Here is a difference of 16.4 per cent. in favour of the vaccinated." Thus, the difference between 46.7 per cent. and 30.3 per cent. is 16.4 per cent. in favour of vaccination; but when something on the opposite side is in question, the difference between 6.4 per cent. and 3.4 per cent. is no less than 46.9 per cent. Previous to giving these striking statistics, there is an endeavour to support the analogy of fever and small-pox by quoting from Dr. Ogle that—

Fever and Small-Pox.

> "It is impossible to make similar comparisons in the case of scarlet fever or measles, and diseases that only affect children. Fever is the only one of the zymotic headings that you can take, because it is the only one that affects all ages to any extent. Fever, is therefore, the only [one] which it is possible to subject to this kind of investigation."

But a reference to the context (Q. 516—8), would have shown that Dr. Ogle was thinking only of "sanitary efforts made of late years"—that is, of a period at whose commencement primary vaccination had already performed the bulk of its task in the way of removing small-pox from child life; so that small-pox, which in pre-vaccination times had been so largely a disease of childhood, had already undergone much change of age-incidence, and had become more easily contrasted with fevers which attack all ages than with measles and scarlatina, which, in the absence of any Jennerian prophylaxis, continue to rage among the young, as they and small-pox did of yore. For those, however, who wish to measure the full effects of vaccination, the comparison must be, not merely between less vaccination, say in 1850, and more vaccination, say in 1880, but also and principally between no vaccination in

the last century, and vaccination practised to the fullest extent to which it has been practised, in the present century. Therefore it is that to compare vaccination and no vaccination, we turn from the present century to the past; and from typhus and typhoid fever to measles and whooping-cough, which, along with small-pox, were the zymotics of childhood before vaccination had come to differentiate among them. And, as to these diseases we find that they continue as before to be specially the maladies of infancy and childhood; while small-pox, as we know, has so far departed from that period of life which is most under the influence of vaccination, that while in Geneva in 1580-1760, of every 1,000 small-pox deaths at all ages, 961 were under 10 years old; in London, in the year 1884, in the vaccinated community the 961 had become reduced to 86, while in the unvaccinated community it stood at 612.

The Benefit to the Unvaccinated.

That there has been some reduction even in the unvaccinated children's share of small-pox has not escaped the notice of the dissentients; and though the statistics which they quote (Section 147) are valueless for the purpose, owing to the "not stated" group altogether overshadowing the other two groups, yet the fact may be freely admitted. The amount of change is, of course, much less than among the vaccinated. The cause of the change is not far to seek. The quality of vaccination is not strained. It blesses even the unvaccinated, by surrounding them with a cordon of the vaccinated, by lessening their chances of exposure to small-pox, and by diminishing the frequency of epidemics. For among the unvaccinated, as in past centuries, the age-incidence largely depends on the intervals between epidemics. While the small-pox share of 961 per 1,000 in 1580-1760 in Geneva had fallen in London in 1884 to 86 among the vaccinated children, it had fallen to 612 among the unvaccinated children; the fall to 86 being directly, and to 612 indirectly, due to vaccination.

While the best and most obvious comparison is between no vaccination in the last century and vaccination in the present century, there is no need to avoid the less striking differences which exist between periods with differing amounts and differing incidence of vaccination. The following Table may be cited:—

From pages cxii.—cxiii. of the Registrar General's *Supplement to the 45th Annual Report.*

Let the death-rate as given at all ages be taken as unity. Then the death-rate for children under five years will be as follows:—

	1851-60.	1861-70.	1871-80.
From All Causes	3.0	3.0	2.9
„ **Small-pox**	**4.7**	**4.0**	**2.2**
„ Measles	6.8	6.8	6.8
„ Scarlatina	4.7	4.7	4.8
„ Diphtheria	4.0	4.1	3.9
„ Whooping-cough ...	7.2	7.2	7.1
„ Fevers (including typhus, typhoid, and ill-defined)	1.5	1.4	1.3
„ Diarrhœa	4.9	5.6	6.1

The Table speaks for itself.

So far, we have been contrasting small-pox with other diseases, with reference to the suggestion that causes other than vaccination (such as sanitary measures) might account for the altered age-incidence of small-pox.

The question whether sanitation is the agency may, however, be put directly to small-pox itself without reference to other diseases. The thesis is that sanitation, having a special influence over the young, may account for the lessened share of children's small-pox. Let us search for the answer in the records of infantile small-pox. Sanitation, being beneficial for tender ages will be beneficial for children under six months old. In Scotland, the vaccination age is six months. If, therefore, in Scotland sanitation be the agency, the fall in the children's contribution to small-pox will appear from the very beginning of life. If, on the other hand, it be vaccination, the fall will show itself only from the age of six months. What are the facts? The Scottish Vaccination Act dates from 1863. In the nine years prior to the Act (1855-63) children under six months old contributed 139 of every 1,000 small-pox deaths at all ages. In the twenty-four years subsequent to the Act, children under six months contributed 138 of every 1,000 small-pox deaths. The proportion was practically unchanged. If the diminution of 1 death per 1,000 (from 139 to 138) be claimed for sanitation, let it by all means be attributed to sanitation. At six months of age, vaccination comes in. In the nine years prior to the Act, the second half-year of life contributed 153 of every 1,000 small-pox deaths. In the twenty-four years subsequent to the Act, the second half-year of life contributed only 47 of every 1,000 deaths from small-pox. The statistics of Scotland, therefore, declare that it is vaccination, not sanitation, which is responsible for the changed age-incidence.*

Small-Pox of Infants in Scotland.

Let us turn now to Germany. Both vaccination and re-vaccination are compulsory in Germany, and small-pox in Germany has undergone much greater diminution than in this country. But opponents of vaccination never tire of

Small-Pox of Infants in Germany.

* *Royal Commission's Report*, vol. vi, App. 636.

attributing the diminution to sanitation. Now it happens that vaccination in Germany is not compulsory at three months as in England, nor at six months as in Scotland, but falls to be performed before the termination of the calendar year following the year of the birth of the child: that is, any time between the age of 12 months and 24 months. This period, say the first two years of life, constitutes the field for the control experiment as between vaccination and sanitation. In England and Wales we have seen that the whole of the first five years of life furnished in 1872-87 only 242 small-pox deaths in every 1,000 small-pox deaths at all ages. But in Germany, the returns of the Imperial Health Office show that in the five years 1886-90, of 735 small-pox deaths at all ages, no less than 301, or 410 per 1,000, were under two years of age.* Germany, like Scotland, declares that the agency against small-pox is vaccination, not sanitation.

Small-Pox of Infants and children in Sheffield and Leicester, etc.

Next, let us appeal to anti-vaccination itself for a verdict. Sheffield, at the time of its small-pox, was well vaccinated (as to primary vaccination), while Leicester was ill-vaccinated. By opponents of vaccination it is declared, on the other hand, that Sheffield was an insanitary town, and Leicester a sanitary town. On the theory of the dissentients, therefore, Leicester ought to have had much less of its small-pox among children than Sheffield had. But the facts are that in Sheffield children under ten years contributed only 25.6 per cent. of the small-pox deaths, while in Leicester they contributed 66.6 per cent. or 71.4 per cent.† The Table containing these data may be a little amplified as follows:—

Percentage of Total Small-pox Deaths borne by Children under 10 Years of Age, in Recent Outbreaks in Various Towns.

Towns.	Vaccination Default.	Percentage of Total Small-pox Deaths borne by Children under 10 Years of Age.
Warrington ...	Very slight	22.5
Sheffield ...	" "	25.6
London ...	In 1883-91, 10 per cent. ...	36.8
Dewsbury ...	In 1882-92, 32.3 per cent. ...	51.8
Gloucester ...	In 1885-94, 10.6 to 85.1 per cent....	64.5
Leicester ...	In 1883-92, 43.8 to 80.1 per cent....	71.4‡

* Mr. Sweeting, in *The Practitioner*, 1892.

† The difference depends on the exclusion or inclusion of three deaths which occurred owing to infection of a scarlet fever ward through proximity to the small-pox hospital.

‡ or 66.6 (see Section 182 *Royal Commission's Final Report*).

As the needle follows the pole, so does increased share of small-pox borne by children follow increased default in vaccination of children.

With regard to the other points mentioned by the dissentients concerning age-incidence, a reply would consist in quotation of the observations on the same points by the Commission in its Report. Further reference, however, is made to the age-incidence of small-pox in "Healthy Districts", at p. 181 of this paper.

RE-VACCINATION.

This subject is dealt with at much length by the Commission, in Sections 299-350 of their *Report*. The dissent of Dr. Collins and Mr. Picton is contained in Sections 149-153. As there is so little to state in the way of Grounds of Dissent, observations on these grounds may be correspondingly brief.

In the first place, it may be convenient to revert to the conclusion of the Commission which is dissented from. It is: "That re-vaccination restores the protection which lapse of time has diminished, but the evidence shows that this protection again diminishes; and that to ensure the highest degree of protection which vaccination can give, the operation should be at intervals repeated."

The first statement made by the dissentients is—

> "It will be seen from the reports made to us that re-vaccination is by no means an absolute protection. At Warrington, of sixty-four re-vaccinated persons living in houses invaded by small-pox, eight were attacked, giving an attack rate of 12.5 per cent.
>
> "In London, of 108 cases of small-pox in re-vaccinated persons, seven were severe, and four, or 3.7 per cent., fatal: a fatality-rate higher than in the once vaccinated class." (Section 149).

But this is not dissent. It is simply selecting the more extreme of certain facts which led the Commission to conclude that protection again diminishes, and that if the highest degree of protection is desired, the operation should be again repeated.

Then in Section 150, as to the Army, the opinion of Brigadier Surgeon Nash is quoted that re-vaccination there "*is* as perfect as endeavours can make it;" and following on this Dr. Collins and Mr. Picton say that: "From the Table he put in we learn that from 1860 to 1888 inclusive

The Army.

there were 3,953 cases of small-pox and 391 deaths in the army, giving a case mortality of 9.9 per cent." Surely, however, in giving small-pox statistics beginning with 1860, the dissentients are putting on the word "*is*," which I have emphasised above, a burden which it has no right to bear. The Minutes of Evidence show that this is so. The order for re-vaccination was first issued on 21st September, 1858. It applied only to recruits, and not to soldiers. At that time the short service system had not begun, and from 1859 it took till about the end of the year 1870 before the same number of men had entered the army as represented its strength in that year. Besides, there were re-entries for longer service up to about 1873 (Q. 3430-3507). If, therefore, the value of re-vaccination is to be measured by the army statistics—and this is the test which the dissentients apparently desire to apply in quoting the figures for the Army—one would expect a very considerable contrast between the earlier and later part of the period 1860-1888. Here is the Table in detail, of which only the totals are given by Dr. Collins and Mr. Picton :—

Once more the figures speak for themselves.

Next, to learn exactly what Dr. Nash meant by re-vaccination in the Army being in 1889 "as perfect as endeavours can make it," it is necessary to note the words which immediately precede those quoted by the dissentients:— "*Q.* 3,559 (Dr. Collins). In the opinion of the Army Medical Department, is the vaccination and re-vaccination of the Army thoroughly satisfactory? *Answer:* It is satisfactory; but, like any other large undertakings, you cannot say that it is absolutely perfect." It appears to me that want of perfection in army vaccination is strongly indicated in Dr. Nash's Table B,* where it is recorded that even in primary vaccination of children the failures in every thousand operations ranged from a minimum of 82 to a maximum of 141 in the years 1882-1888. Such a proportion of failures is simply unknown in the practice of public vaccinators among the civil population, and is calculated to raise considerable suspicion as to the still larger percentage of failures in the Army practice of re-vaccination.

The dissentients go on to show that the amount of small-pox in the army varies at different places and times; and they show that in Assouan, in 1888-89, when the disease prevailed among the native population, it broke out also among the troops, and that when the troops were less

* P. 277, *Royal Commission's Report*, Vol. ii.

exposed to infection they were less infected. It is not easy to see the point of this. For of course any susceptibility to small-pox which may remain in the Army, owing to failures or imperfect results of re-vaccination, or from whatever cause, will show itself according to the amount of opportunity for infection.

TABLE *showing the Average Annual Strength of the British Army, the Number of Admissions and Deaths from Small-Pox, together with the Ratio per* 1,000 *for the Twenty-nine Years*, 1860-88

Year.	Average Annual Strength.	Admissions.	Deaths.	Admissions.	Deaths.	Remarks.
1860	203,017	329	30	1.6	.15	
1861	195,281	331	43	1.7	.22	
1862	196,349	184	20	.9	.10	
1863	196,339	228	18	1.2	.09	
1864	191,057	325	36	1.7	.19	
1865	184,503	270	27	1 5	.15	
1866	175,303	101	7	.6	.04	
1867	174,614	109	6	.6	.03	
1868	173,031	153	3	.9	.02	
1869	163,905	176	18	1.1	.11	
1870	155,945	66	9	.4	.06	
1871	171,455	329	36	1.9	.21	
1872	171,956	191	24	1.1	.14	
1873	169,744	98	13	.6	.08	
1874	169,014	57	8	.3	.05	
1875	169,235	20	3	.1	.02	
1876	169,197	45	2	.3	.01	
1877	174,884	72	6	.4	.03	
1878	185,006	89	15	.5	.08	
1879	164,642	39	3	.2	.02	32 attacks and 3 deaths in India
1880	159,622	14	2	.1	.01	12 attacks in India
1881	173,331	38	3	.2	.02	23 attacks in United Kingdom ; 15 in India
1882	174,557	64	5	.4	.03	Epidemic in India (44 attacks)
1883	168,383	126	12	.7	.07	Epidemic in India (100 attacks, 84 in Bengal alone)
1884	167,686	114	9	.7	.05	Epidemic in India (77 attacks), in Egypt 35 attacks
1885	177,928	86	7	.5	.04	Egypt, 52 attacks ; India, 12 ; United Kingdom, 19
1886	188,739	84	4	.4	.02	Egypt, 51 attacks, 3 deaths ; India, 22 attacks and 1 death
1887	194,037	45	7	.4	.04	Epidemic in India (40 attacks), Egypt (26 attacks)
*1888	199,567	140	15	.7	.07	Epidemic in India (10 attacks), Egypt (14 attacks)

* Approximate.

Small-Pox Nurses.

The dissentients do not deny (Section 151) that re-vaccinated nurses exhibit remarkable immunity from small-pox; and if it be true that nurses have sometimes shown immunity, even in the absence of re-vaccination, it accords with the view that primary vaccination has now and again a protective power of very lengthened duration, even against attack by small-pox. With regard to the Bicêtre Hospital where it is said (Section 152), that not one of a medical and nursing staff of eighty persons was attacked by small-pox, though "so many" of them had not been re-vaccinated, there is nothing to indicate how many are included in the "so many," nor whether their primary vaccination had been performed in infancy or later, nor what were their ages at the time of the outbreak. The experience of the Leicester nurses (not quoted by the dissentients) differed sadly from that of Bicêtre, and the figures are quite accurately known. The staff amounted to forty, of whom twenty had been vaccinated or re-vaccinated shortly before beginning duty, fourteen had had small-pox or been re-vaccinated at some previous time, and six refused re-vaccination. Among the thirty-four (20 + 14) there was one mild case of small-pox, in a person whose re-vaccination had been done ten years before. Of the six who refused re-vaccination, only one now requires any protection against small-pox. The other five were seized by the disease, and one of them died. There is ample experience that while re-vaccinated nurses do not take small-pox, fever nurses have very often taken fever; so that in some hospitals only those were safe who had already had fever; and the speculative view that repeated exposure without attack gives protection (Section 152) need not detain us, especially as the dissentients do not themselves venture to accept it.

In Section 151 they say:—

> "While some hold that an unsuccessful re-vaccination is of no account, others, in accordance with the teaching of Jenner and Bryce, regard it as indicative of insusceptibility, and assert that as long as a person is liable to successful vaccination he is liable to take small-pox; and that, therefore, insusceptibility to re-vaccination indicates protection."

We have already seen that Bryce's test was performed within three or four days of vaccination, and had no bearing on the question of duration of protection; while as to re-vaccination, the dissentients must be well aware that a person who is unsuccessfully operated on one week is often

successfully re-vaccinated the next week, and that it is therefore quite impossible to look on failure of the operation as equivalent to insusceptibility to small-pox. Dr. Gayton's experience that well-vaccinated nurses and servants may have trifling illnesses—sore throat and the like—possibly due to small-pox infection, has its parallel in the experiences of previous centuries, that small-pox nurses who had had small-pox got occasional pimples and the like in the course of their duties; and similar facts might be cited with regard to scarlet fever in the present day.

The evidence of the protective power of primary vaccination performed in infancy is generally applicable to the protective power of the operation when repeated in later life. The contrasts which have been found to exist between susceptibility to small-pox amongst the unvaccinated and the vaccinated in the early periods of life, hold good between the once vaccinated and the re-vaccinated in later years. In a stricken town the contrast between the amount of small-pox in the postal and police services, the military, the nurses in small-pox hospitals, and the employès in public works where re-vaccination is enforced, on the one hand, and among those sections of the community which have to trust solely to a vaccination performed in infancy on the other hand, is invariably a striking contrast; and every fresh outbreak of small-pox, even in communities like Leicester and Gloucester, where primary vaccination has been most neglected, furnishes a fresh illustration of the value of repeated vaccination.

I do not propose to recapitulate the abundant evidences of the protective power of re-vaccination contained in the Report of the Commission.

The Relationships of Vaccinia and Variola.

This and related questions are discussed in Sections 154-176. So far, in reviewing the Grounds for Dissent, I have been dealing mainly with questions of fact, and with inferences from fact. Now, however, a much more speculative and recondite subject is entered upon, in which there would be little profit in following every detail, and in setting up one opinion against another on point after point. Accordingly, I shall glance only very shortly at the Grounds of Dissent as here set forth. Experiments in variolation of the cow are first referred to. It is alleged (Section 154) that "in none of the experiments have the usual signs of natural cow-pox been found to result." This

refers apparently to the inflammation and irritation, and so forth, which Ceely ascribed to the "merciless manipulations of the milkers." In the absence of such manipulations, such "signs" should not show themselves. They do not belong to the phenomena of vaccinia as seen in calves on whose skin lymph is cultivated, and they ought not to appear as a result of variolation in the absence of constant abuse of the parts. After reference to Chauveau's experiments, the attempts to attenuate small-pox made by Thiele and Adams are again mentioned (see p. 105, *ante*); and it is stated that "Dr. Walker, who carried on a large vaccination practice in London, in the beginning of the century, appears to have entertained similar views, and practised the dilution with water of the small-pox virus." To prevent misapprehension, it has to be noted that Dr. Walker does not speak of having practised anything alleged to be *vaccination* with this material, though, as mentioned, he had a large vaccination practice.

Dr. Walker.

Prof. Crookshank's evidence is next referred to, in support of the view that certain other agencies can produce vesicles not greatly differing from vaccine vesicles, or from those recorded by Adams in his experiments with a "sport" of small-pox. The purpose of citation of evidence of this sort is to cast doubt on the view that because what are in every respect undistinguishable from vaccine vesicles can be raised from human small-pox (through the intervention of the cow), "there is therefore any special or essential inter-relation between cow-pox and small-pox." The value of this evidence for the purpose in question must be matter of opinion, and I do not propose to discuss the weight to be attached to the conclusions of the dissentients on the one hand, and of the Commission on the other. It appears, however (Q. 11,542-3), that as regards syphilis, Professor Crookshank was rather engaged in explaining to Dr. Collins and the Commission why the local result of syphilitic inoculation does *not* resemble vaccine vesicles, than in asserting that it does. It seems not to have struck the Commission that much explanation was needed here.

"Spontaneous" Cow-Pox.

Believing as he did that all true cow-pox was derived from horse-pox, Jenner conveniently described as "spontaneous" other forms of eruption arising on the teats of cows, and in this sense he used the word "spontaneous" as equivalent to spurious. This gives no ground for arguing that cow-pox is not genuine because it may not have been transmitted to the cow from the horse. Jenner's use of the

word "spontaneous" as equivalent to spurious had its origin in his theory about horse grease, but cow-pox and horse-pox may be identical without all cow-pox having had an equine source. Blister pock, white pock, yellow pock, &c., are referred to by Ceely as spurious pocks, but neither he nor Klein suggests that they have been used for vaccination; and I am not aware that it has been proved that they can produce vesicles of any sort capable of transmission from arm to arm (Sections 164-7—170).

If the promises of success in the bacteriological investigation of variola and vaccinia are fulfilled, it may be possible bacteriologically to meet the demands of opponents of vaccination for definitions, but probably even then they would prefer to continue to describe vaccination as "a grotesque superstition," though the author of this description himself quoted, not inappropriately, John Hunter's saying that definitions are, "of all things on the face of the earth the most cursed." Meanwhile, where small-pox prevails, protection by vaccination may serve as a substitute for definition of vaccination, and we may continue to employ the telegraph and the telephone in the notification of cases without worrying about the exact nature, and description, and definition of electricity (Section 168).

In Section 169, regarding the application of the variolous test to Ceely's lymph derived from small-pox inoculated of the cow, the papulo-vesicular elevations described by Ceely would not have been looked on as evidence of susceptibility to small-pox if the tests had been applied to variolations in the last century, before vaccination was heard of.

In Sections 173-176, Pasteur's inoculations for anthrax and pleuro-pneumonia are discussed, and mention is made of anti-cholera inoculations, but these subjects need not be entered on here.

Alleged Injurious Effects of Vaccination.

Sections 177-220 are devoted to this subject. In Section 177 we read:—

> "The Select Committee of 1871 reported 'that if the operation be performed with due regard to the health of the person vaccinated, and with proper precautions in obtaining and using the vaccine lymph, there need be no apprehension that vaccination will injure health or communicate any disease.' Even more recently this view has been reaffirmed in a pamphlet, entitled, *Facts Con-*

cerning Vaccination for Heads of Families, 'revised by the Local Government Board, and issued with their sanction,' which states that 'as to the alleged injury from vaccination, all competent authorities are agreed that, with due care in the performance of the operation, *no risk of any injurious effects* from it need be feared.'

"We agree with our colleagues that, notwithstanding repeated and emphatic assertions to the contrary, the admission must without hesitation be made that risk attaches to the operation of vaccination."

After all, is there much difference between the finding of the Select Committee and the statement in the Local Government Board pamphlet on the one hand, and the conclusion of the Royal Commission on the other hand? The Select Committee say, "there need be no apprehension" of injury. The pamphlet says, "no risk . . . need be feared." And the Royal Commission says that the dangers "are insignificant," and that there is reason to believe that even these insignificant dangers "are diminishing," and "will do so still more in the future." One need not fear, nor be apprehensive of, dangers which are both insignificant and diminishing.

Diminishing Risks.

Since vaccination was introduced, the evils which have been attributed to it by one individual or other, and at one time or another, have been very numerous, but of recent years the list even in anti-vaccination literature has been enormously curtailed. Of alleged dangers some fifty or more can easily be culled from anti-vaccination literature since the beginning of the century, but the great majority are seldom heard of nowadays. In their report on this part of the subject, the Commission begin by the consideration of fourteen diseases, those namely which formed the subject of a Parliamentary return obtained in the year 1877 by Mr. C. H. Hopwood, Q.C., who was also a witness before the Commission. This Parliamentary return they discuss as follows:—

Mr. Hopwood's Parliamentary Return.

"383. The first six of these diseases showed an increasing, the next four a decreasing, mortality, whilst the remaining four exhibited an irregular mortality, there being in three cases an increase in the second period and a decrease in the third, and in another case a decrease in the second, but a slight increase in the third, when, however, the mortality was not so high as in the first period.

When all the diseases were taken together, there appeared to be in the aggregate an increasing mortality. Some found in this circumstance evidence of the malign influence of vaccination. Such a conclusion is manifestly untenable. There was no more reason for attributing to vaccination the increase of mortality in the case of those diseases where the mortality had grown, than there was for asserting that to its beneficent influence was due the decrease of mortality in those cases in which the mortality had become less. The hypothesis that it caused the mortality in some instances to grow, and in other instances to decrease, and that it was responsible for the balance of increase shown on an aggregation of the two, does not merit serious attention. It is not as if all the diseases in the class showing an increasing mortality were such as could be deemed capable of being affected by vaccination, whilst those included in the class with a decreasing mortality were in a different category. Two of the diseases included in this latter class, viz., convulsions and pneumonia, have been regarded in particular cases, even on recent occasions, as having had their origin in vaccination."

A diminished List.

In 1885, Dr. Alfred Russel Wallace made, not fourteen but only five claims as to vaccinal evils :—syphilis, cancer, tabes mesenterica, pyæmia, etc., and skin disease. From this list Dr. Collins and Mr. Picton have dropped cancer, while tubercular disease is only doubtfully charged against vaccination. Syphilis, erysipelas, septic infection, skin eruptions, and alleged exceptional results of vaccinia itself, etc., include the more specific accusations in the Statement, though there is also mention of leprosy, and as just noted, of tubercle. This covers quite a wide field of allegation, yet the list is greatly shrunken compared with lists of even fifteen to thirty years ago. Various accusations brought against vaccination during the sitting of the Select Committee of 1871 have quite fallen out of sight. It is most important, however, that every untoward event, no matter how trifling, which may have any share of its cause in vaccination should receive the fullest and most candid consideration; and in the course of their seven years' labours, the Commission have done no better work than in following up, through their medical staff, every case of

injury or alleged injury that could be heard of anywhere in England or Wales. The assertions of violent opponents of vaccination, when traced to their source, are so constantly found to be baseless that there may be a tendency on the part of the medical profession to assume too readily that vaccination is always and everywhere absolutely harmless. It is the duty of every medical man to resist this tendency. The reports of the medical men appointed by the Commission to investigate cases of injury, or supposed injury, should be carefully studied, especially by every public vaccinator, so that he may make himself fully acquainted with even the remotest possibilities of harm.

Seeing that Dr. Collins and Mr. Picton hold the anti-variolous power of vaccination to be less and the possible usefulness of compulsory isolation to be greater than the Commission believe, it is natural that they should attach more weight than do the Commission to any suggestions of mischief accompanying or resulting from vaccination. Hence they proceed (Section 178), to recite some of the findings of the Commission, and then to declare (Section 179):

> "Putting together all these admitted elements of danger, though each may be slight in itself, we think that the sum of them constitutes a very serious objection even to the form of modified compulsion favoured by our colleagues."

In Section 180 they write:

> "We cannot understand on what principle a parent . . . is not entitled to refuse the not unreal risks of calf lymph, though he also regards these with abhorrence."

As to this last point, I do not understand the attitude taken up by the dissentients. They surely for the moment have forgotten that the Commission *does* propose to entitle every parent to refuse even calf lymph, and all that it insists on is that some effort be made by the State to ascertain that a parent really does regard the alleged risks with abhorrence, or even with such degree of opposition short of abhorrence as may be called "conscientious objection." The dissent here seems to be based on failure to grasp the meaning of a very plain statement made by the Commission.

Section 184 gives a pageful of statistics to show that the risks of railway journeys are much less than those of

vaccination; but while each vaccination lasts for a fortnight or more, an average railway journey may be a matter of an hour or less.

Erysipilas and Syphilis.

Sections 185-200 deal with erysipelas, and contain extracts from the reports of the medical men appointed by the Commission to inquire as to injuries, and from the reports of the medical department of the Local Government Board.

The dissentients cite instances in which erysipelas apparently had its origin in vaccination, without mention being made of vaccination in the death certificate. Their comments lead up to the view that erysipelas is not infrequently related to vaccination itself, and is so far unpreventable (Section 198). The Commission, on the other hand, discuss the subject with regard to possible methods of prevention. They believe that in cases of erysipelas subsequent to vaccination, the poison is often extraneously introduced and might often be avoided. They point out that erysipelas is a very frequent disease of childhood: nearly two thousand per million dying of it during the first three months of life—so that the weeks immediately following vaccination cannot in any event be expected to be exempt from erysipelas, and that where both vaccination and erysipelas are mentioned in a death certificate, there is not necessarily any causal connection between them.

Regarding syphilis, Section 202 says:

> "We agree with our colleagues that the possibility of vaccine syphilis, formerly denied, has been fully established."

The only medical man whom I know of as likely to disagree with this verdict is Dr. Creighton, who holds that so-called vaccinal syphilis is "in no respect of venereal origin."* Dr. Collins and his colleague state that "safeguards which have been laid so much stress on are now known to be illusory." If with regard to humanized lymph (which is here in question), every safeguard which is not an absolute bar to syphilis can be described as "illusory," then the assertion need not be disputed, but the English language is not usually so employed. Care in selection of vaccinifers of healthy appearance and with typical vaccine vesicles; care in the taking of lymph on the eighth day and only as it flows freely from the vesicle; a knowledge of the family history of the child, aided by the avoidance of first-born children; these precautions are not "illusory."

* *Cow-Pox and Vaccinal Syphilis*, 1887, pp. 124-5.

The Commission, indeed, declares that "where the antecedents of the vaccinifer are fully ascertained, and due care is used, the risk may for practical purposes be regarded as absent."

It is a suggestive fact that the great bulk of evidence of the possibility and actuality of vaccinal syphilis comes from abroad. The difference is more than geographical. The subject under discussion by the Commission is vaccination as practised with the degree of care and caution which is exercised in this country, and not as described in connection with foreign blunders: as, for example, where we find tenth-day lymph used, and such numbers as 46 and 56 vaccinated from a single vaccinifer whose vesicles must obviously have been drained (with consequent exudation of blood serum), to an extent that would never be dreamt of here.

Unconscious Memory.

While the dissentients are absolutely opposed to Dr. Creighton's view that so-called vaccinal syphilis has nothing to do with venereal disease, they make much of his opinion that vaccination itself may occasionally take on a character and have results closely resembling the phenomena of syphilis. This doctrine of Dr. Creighton's has its origin in his theory of "unconscious memory in disease," formally promulgated in 1883. He looks on sore arms following vaccination in the present day as acts of memory on the part of cow-pox, as reversions perhaps to the sore on some cow's teat or milker's hand, which had given rise in the beginning of the century to a stock of lymph carried on through thousands of removes from then till now. Regarding erysipelas, the dissentients say in Section 197:—

> "We learn from bacteriological investigations that vaccine lymph contains a great variety of germs or micro-organisms, some of which are accounted to be pathogenic or disease-producing; and though none of them have been identified as the active principle of the vaccine disease, it seems clear that in some specimens germs believed by high authorities to be those of erysipelas have been encountered."

Dr. Creighton, however, will have nothing to do with such bacteriological theories. He looks on erysipelas, not as due to any of Professor Crookshank's germs, but as being another act of "memory," and the word memory, he insists, is not used analogically, but in its everyday meaning. Dr. Creighton's doctrine is quite opposed to that of Pro-

fessor Crookshank. Both, however, lend themselves to opposition to vaccination, and the dissentients call on both to testify doctrinally against vaccination, making no note that their doctrines testify against each other.

I have elsewhere* reviewed at some length Dr. Creighton's thesis, and do not propose to make further reference to it here.

In Section 215 we read:—

Auzias-Turenne.

"This view of the affinity and results of cow-pox is that which was foreshadowed in the writings of Auzias-Turenne, and which in this country has been chiefly advocated by Dr. Creighton."

As the name of Auzias-Turenne has been repeatedly cited in support of the allegation that cow-pox presents some kind of analogy to syphilis, it may be proper to quote from Professor Crookshank's translation† a passage, the bearing of which seems not to have been observed.

"In my studies of the virulent maladies, I am accustomed to take syphilis as a type and a guide. As it progresses by definite stages, its course can be observed. The phenomena which I observe in this malady I endeavour to detect in the evolution of other diseases. Here they are much more rapid, and, in consequence, much less apparent; nevertheless, I nearly always succeed in discovering them. I assure myself, in every case, of their reality or of their absence; and this verification constitutes knowledge—a scientific conquest. To my mind, he who subordinates the study of other virulent maladies to that of syphilis, may be compared to a traveller who, proceeding by short stages, has time to take cognizance of the country through which he passes, instead of traversing it without observing anything—by rail. However rapid, therefore, the virulent maladies may be in their course, their stages may be traced with the help of syphilis, which progresses slowly. Analogy is a valuable aid, but it must be carefully employed. It is an adventurous guide, whose directions must be kept under a firm control."

Seeing that Auzias-Turenne takes syphilis as a "type and guide" of all virulent maladies, his views have to be read accordingly, and his value as a witness has to be weighed accordingly. But if those who have been in the habit of citing him really want to profit by his writings

* *Edinburgh Medical Journal*, 1889-90.

† *History and Pathology of Vaccination*, vol. ii, p. 555.

they should give particular heed to the closing sentences in the above passage: "Analogy is a valuable aid, but it must be carefully employed. It is an adventurous guide, whose directions must be kept under a firm control."

Infantile Syphilis in Scotland.

In a concluding paragraph (Section 216) the dissentients admit that the increase in registration of deaths from syphilis, shown by the Registrar-General's reports, cannot be set down to vaccination, and then they make a reservation as to the possibility of much syphilis looked on as hereditary being really vaccinal. But the reservation might as well have been omitted, for it is not suggested that any disease can be attributed to vaccination in children on whom vaccination has not been performed. In Scotland the vaccination age is six months. In the nine years (1855-63) preceding compulsory vaccination in Scotland, the second half-year of life contributed 109 of every 1,000 deaths from syphilis at all ages. In the periods 1864-75 and 1875-87, when vaccination at six months had become obligatory, the corresponding contributions were 118 and 109, or almost the same as before. Contrast these figures with those of the first half-year of life, in which deaths from vaccinal syphilis cannot be alleged. In the nine years preceding compulsory vaccination the contribution was 575 to every 1,000 syphilis deaths registered for all ages. In the periods after vaccination at six months of age had become compulsory, the babies under this age, whom the new law did not take cognizance of, contributed 612 and 647 deaths respectively in every 1,000 deaths from syphilis at all ages. Thus not only do the unvaccinated babies account for nearly two-thirds of all deaths from syphilis, but their share has appreciably increased; while that of the children in the second half-year, being the first period of life under the influence of vaccination, has all along been much less than that of the younger babies, and in 1875-87 was just the same as it had been in 1855-63, notwithstanding the interposition, in 1863 of the new law of vaccination.

"Vaccinal" Diseases in Leicester.

I have omitted to notice the remarks which Mr. Picton and his colleague make regarding some Leicester statistics of so-called vaccinal diseases, put in by Dr. Ogle, the Superintendent of Statistics in the Registrar-General's Office. The statistics themselves are not quoted, but as Leicester and its experiences since it gave up vaccination are constantly adduced in support of anti-vaccination, the Table may be given here for the better understanding of the question:—

Deaths under One Year of Age from Certain Causes per Million Births, in Leicester and in England and Wales, in the Periods 1863-67, 1873-77, *and* 1883-87 ; *and Increase or Decrease per Cent. in the Twenty Years.*

Period.	Diarrhœa and Dysentery.		Syphilis.		Erysipelas.	
	Leicester.	England and Wales.	Leicester.	England and Wales.	Leicester.	England and Wales.
1863-67 ...	42,627	14,908	988	1,511	680	750
1873-77 ...	48,594	15,975	1,371	1,754	1,724	878
1883-87 ...	44,409	14,983	1,673	1,884	962	625
Increase or Decrease (–) per cent. in 20 years	4.2	.5	69.3	24.7	41.5	–16.7

TABLE—*continued.*

Period.	Tabes Mesenterica and Scrofula.		Bronchitis.		All other Causes.		All Causes.	
	Leicester.	England and Wales.	Leicester.	England and Wales.	Leicester,	England and Wales.	Leicester,	England. and Wales.
1863-67 ...	4,819	4,553	9,452	10,458	153,580	122,554	212,146	154,734
1873-77 ...	6,013	5,418	12,734	15,922	140,565	108,338	211,001	148,285
1883-87 ...	6,064	5,772	20,114	18,125	132,892	101,770	206,114	143,159
Increase or Decrease (–) per cent. in 20 years	25.8	26.8	112.8	73.3	–13.5	– 17.0	–2.8	–7.5

The observations of the dissentients regarding this Table are contained in Section 181.

"We are not prepared to attach much weight to figures put in by Dr. Ogle, instituting a comparison between Leicester and the whole of England and Wales, in regard to the changes in the infantile mortality from various diseases.

To make such comparison valuable it would be, as Dr. Ogle seemed inclined to admit, better to compare an urban population similar to that of Leicester, but in which vaccination was thoroughly carried out. If we want to ascertain by the method of differences whether vaccination exerts a detrimental effect by increasing the mortality from certain infantile diseases, it is surely imperative to see that the places or times compared differ as little as possible in respect of circumstances other than vaccination.

(Section 182), "In the statistics which Mr. Biggs furnished, we do not find any evidence that the increasing disuse of infantile vaccination in Leicester has prejudicially affected the mortality of young children; on the contrary, there has not only been a marked reduction of the general death-rate since 1875, but a reduction in the death-rate of infants under one year, a rate which reached its highest point since 1838 in the period 1868-72, when vaccination was most thoroughly enforced."

But the dissentients know very well that Dr. Ogle did not put in his Table with any view of showing that it was vaccination which increased the mortality in Leicester from the tabulated diseases. He was quite aware that vaccination had greatly diminished in Leicester, while diseases often attributed to vaccination by its opponents had greatly increased in Leicester. Nor did Mr. Biggs, who is the leading anti-vaccinationist in Leicester, attempt to furnish evidence that diminishing vaccination had caused an increase in the general death-rate of infants. The objection that Leicester is contrasted with England in the Table, and not with a large town, is replied to by pointing out that the Table refers to three successive periods of time in which Leicester is contrasted with Leicester, and England with England.

The meaning of the statistics is not far to seek, and need cause no perplexity. The list of alleged vaccinal diseases is constantly changing. So long as a disease attributed to vaccination continues to increase, it is apt to remain on the list of vaccinal evils; but when it begins to diminish it is likely to be quickly dropped, or rather to be used in exemplifying the contention that, as this disease fell without any artificial preventive like vaccination, so might small-

pox have fallen without vaccination. Cholera, leprosy, enteric fever and scarlet fever have all served at one time to illustrate, in their increase, the evils of vaccination, and at another time, in their decrease, the needlessness of vaccination. Among the maladies which have been less subject to such fluctuations, and which have with more or less persistency continued to be attributed to vaccination, there are included diarrhœa, syphilis, tabes mesenterica, and scrofula and bronchitis. It may be noted in passing, that the history of bronchitis as a vaccinal malady is interesting. In Mr. Hopwood's first parliamentary return both bronchitis and pneumonia were included; but the statistics obtained in this way showed that while bronchitis was rapidly increasing as a registered course of death, pneumonia was steadily decreasing. A new return was accordingly obtained by Mr. Hopwood, and this time pneumonia was dropped out of the list, while bronchitis was retained. Some other registered causes of death were similarly dealt with.

The result is that the above diseases, along with erysipelas, may still be looked on as among the diminishing list of alleged vaccinal evils. Along with this, there is the fact that Leicester, during recent years, has more and more neglected infantile vaccination, so that infants there are now practically free from any mischief that vaccination can do. This being so, it appears to have struck the Registrar-General that the Vaccination Commission would be interested to know how infants in Leicester had fared with regard to these diseases since vaccination fell into desuetude.

The results are given in the Table, and are sufficiently striking. The vaccinal disease diarrhœa has increased among the unvaccinated infants in Leicester by 4.2 per cent. The vaccinal disease syphilis has increased by 69.3 per cent. The vaccinal disease erysipelas has increased by 41.5 per cent. The vaccinal diseases tabes and scrofula have increased by 25.8 per cent. The vaccinal disease bronchitis has increased by 112.8 per cent. It was open to urge, however, that among the vaccinated infants of England these vaccinal diseases had increased still more, and so the facts for England had to be elicited. And in every one of the diseases, as well as in all deseases combined, England was better off than Leicester. There is no allegation that "the increasing disuse of infantile vaccination in Leicester" has been responsible for this increase. But the figures indicate how much attention need be

paid to the parliamentary returns obtained by statisticians like Mr. Hopwood, Q.C., and Mr. Channing, M.P.

The next subjects dealt with (Sections 221-273) come under the reference—

"MEANS OTHER THAN VACCINATION FOR DIMINISHING THE PREVALENCE OF SMALL-POX."

This at once brings us back to sanitation and Haygarth. The dissentients say:

> "In speaking of sanitation we use the word in its widest sense; we are not speaking merely of drainage improvements, but we include the prevention of overcrowding on areas or within houses and rooms, the proper construction of dwellings, so as to permit thorough ventilation; the promotion of cleanliness by adequate water supply, and the prompt removal of filth accumulations. Related to these measures, but in a somewhat different category, are means directed against contagion, the speedy separation (in suitable hospitals) of the infected from the healthy, the disinfection of persons and things, and the prevention of the propagation of the disease by inadvertent carelessness or by intentional inoculation." (Section 222.)

This is preceded by the statement that:—

> "We have already given our reasons for thinking that the teaching of the early sanitarians, like Howard and Haygarth towards the close of last century, initiated a new line of thought in the prevention of disease; and we believe the general improvement of the public health which then set in was due, in a large measure, to a greater sanitary activity; and that the falling off in the death-rates of fevers and small-pox, as well as in the general death-rate, is confirmatory of this view." (Section 221.)

The value of Howard's philanthropic work with regard to the condition of prisons is universally recognised, but sanitation was with him rather an incident than a leading motive, and we do not find that in the last century prisons were specially associated with small-pox. Jail fever was not variola. Haygarth we have already spoken of as a

"sanitarian," but how does he get included in the term as used by the dissentients? I have argued that for different zymotic diseases sanitation has different meanings; and, in the present day, for small-pox sanitation means vaccination (including re-vaccination), with isolation as a useful auxiliary, other measures coming in a long way behind. So in the last century, for small-pox, sanitation meant, for the individual, inoculation, while isolation as outlined by Haygarth meant very little. The assertion of a commencing influence of sanitation might be made regarding almost any period from the thirteenth or fourteenth century onwards; and, excepting that vaccination, which is for small-pox by far the greatest of all sanitary measures "in the widest sense", began with the beginning of the century, there is no special reason for fixing this time, rather than almost any other previous or subsequent time, as the beginning of "greater sanitary activity."

I hardly understand Section 223, in which the opinion appears to be advanced that the extension and severity of small-pox depends upon "influences other than contagion;" unless all that is meant be that dirt favours contagion, and that propinquity of individual to individual makes contagion conveyable more easily from individual to individual. All the same, the quotation given from the Report of the Royal Commission on Infectious Hospitals does not justify the use of the word "insignificant" as applying to their opinions regarding the comparative importance of hospital influence.

> "We agree," say the dissentients in Section 224, "with the epidemiologist Hirsch that small-pox, as well as typhus, takes up its abode most readily in those places where the noxious influences due to neglected hygiene make themselves most felt."

Hirsch being in question, what he thinks of vaccination may be noted:*—

Hirsch on Vaccination.

"However rich the medical and historical literature may be in accounts of the occurrence of small-pox at various parts of the globe; however clearly the past may bear witness to the amount of human life that has been offered up a sacrifice to this murderous disease, beside which the loss through the bloodiest of wars, or through other severe pestilences such as plague and cholera, appears to be infinitesimally small; and however sufficient may be the researches into the present geographical distribution of the disease,—still the materials available to us are not even in a measure adequate for drawing up a proper history of the pestilence in past centuries. It was not its rarity but, as Häser justly remarks, actually its everyday occurrence, that weakened the

* Sydenham Society's Translation.

interest of epidemiographists towards it. But we may be all the more easily consoled for this defective state of the epidemiological record of small-pox in the pre-vaccination era, by the reflection that even a complete narrative of the small-pox epidemics of that time would have no further scientific interest than as enabling us to vindicate the importance of vaccination for preventing the disease, or, at least, for reducing the mortality caused by it. So far as answering *that* question goes, the materials at our service are amply sufficient; and the question has been already answered so often and so thoroughly, particularly in the classical reports of the English Board of Health under the editorship of John Simon, and in the excellent work of Bohn, that I find no occasion to adduce once more the historical proofs relating to it.

"That the achievement of Jenner was at once a turning-point in the history of small-pox, and a new era in the physical welfare of mankind; that the power of the pestilence became more and more restricted both in range and in severity in proportion as the practice taught by him obtained acceptance and careful attention at the hands of various nationalities; that the disease at the present day, as is abundantly shown in the foregoing account of its geographical distribution, still bears, in those regions where ignorance and prejudice have opposed the adoption of vaccination or where the carelessness of the authorities has neutralised its good effects, the same character for destructiveness that meets us in the medical and chronological accounts, and in the mortality statistics, of European countries in the pre-vaccination period; that even to-day we find in the devastation of populous districts, and in the uprooting of whole tribes, the indications of what this ravaging pestilence could do—all this is so thoroughly brought out in the writings I have named, that it can be only folly or stupidity that would seek nowadays to minimise or to question the immortal merits of Jenner."

Chadwick.

Sir Edwin Chadwick is next quoted (Section 255) as holding that complete sanitary measures would be "the effectual preventive of diseases of those species"—small-pox, typhus, and other "ordinary epidemics." Everyone who knows aught of public health regards with gratitude the splendid labours of Chadwick, but he was going just a little out of his sphere in making a sweeping assertion involving an assumption of knowledge of the possibilities of infectious diseases which no medical man, let alone a layman, can claim to possess.

Small-Pox in India.

Indian experiences of the power of small-pox epidemics to overleap the defences of vaccination are quoted in Section 227. Other experiences of the remarkable power of vaccination to prevent small-pox in India might have been quoted from the writings of Brigade-Surgeon Pringle, of the Sanitary Department of the North-West Provinces and Oudh. I am not clear, however, as to the exact point of these references to India. We have been told, at the very beginning of the Statement, that small-pox has "as it were, its habitat" in India and Central Africa, and that "its diffusion thence results from importations under favourable circumstances of the morbid poison by infected persons and things;" and that in India there are "native foci" of the disease. If all this be so, how do the facts regarding small-pox in India apply to England? And

if small-pox in its native habitat ("as it were") can overpower vaccination, might it not also overpower isolation and "sanitation in its widest sense"? The Commission is dealing with the prevention of small-pox, not in India but in England. And if India and Nubia and the Soudan (Section 228) are native habitats of small-pox, what matters it whether small-pox inoculation is still practised or not?

Social position in addition to sanitary environement is alleged (Section 229) to have a potent influence both on the prevalence and fatality of small-pox. It certainly is the case that small-pox nowadays does not attack or slay many members of royal families or of the highest social circles. But are they not all vaccinated, and are not very many of them re-vaccinated? In times when vaccination was unknown, how did it fare with such dignitaries? Simon answers the question. "In the circle of William the Third, for instance, his father and mother died of it, and not least, his wife; and his uncle, the Duke of Gloucester, and his cousins, the eldest son and the youngest daughter of James the Second; and he himself (like his friend Bentinck) had suffered from it most severely, barely surviving, with a constitution damaged for life. Or again, in the Court of Austria: 'Joseph the First (says Vehse) was carried off, when not more than thirty-three years of age, by the small-pox; to which, in the course of the eighteenth century, besides him, two empresses, six archdukes and archduchesses, an elector of Saxony, and the last elector of Bavaria, fell victims.' To this list might have been added, no doubt, many other names; among them, for instance, a dauphin (1711) and a king (1774) of France, a queen (1741) of Sweden, and an emperor (1727) of Russia."* Social Position.

The dissentients continue:—

"Dr. Farr, before the days of compulsory vaccination, pointed out the effect on causes of mortality of

* *Papers on Vaccination*, 1857. In the *Edinburgh Medical Journal* for June 1871, Dr. John Gairdner, F.R.C.S.E., has a very interesting note on this subject. The following are the deaths from small-pox, with the dates, mentioned by Dr. Gairdner:—Henry, Duke of Gloucester, 1660; Mary, daughter of Chas. I, 1660; Charles, Duke of Cambridge, 1667; Mary, Queen of England, 1694; Maria Louisa Teresia, daughter of James II, 1712; Louis, son of Louis XIV, Dauphin, 1711; Louis, Duke of Burgundy, 1712; the Dauphiness his wife, 1712; the Duke of Bretagne, their son, 1712; Louis XV, 1774; Joseph I, Emperor of Germany, 1711; Peter II, Emperor of Russia, 1730; Henri, Prince of Prussia, 1767; Maximilian Joseph, Elector of Bavaria, 1777.

Among those who had the disease and recovered from it were Queen Anne, Peter III of Russia, and Louis XIV of France.

the selection exercised by insurance companies. Death from the eruptive fevers among such selected lives was rare; among 4,095 deaths in the Equitable Society, during the years 1801-32, only one was from small-pox."

But how many among the insured were vaccinated without compulsion, and among those who were not vaccinated how many had had small-pox and other eruptive fevers before they reached the age for insurance?

The Norwich Epidemic of 1819.

"In the Norwich epidemic of 1819, Cross noted that the epidemic was almost exclusively confined to the very lowest orders of the people."

Cross also says that though vaccination had been offered to the poor, "Such, however, is the indifference of the uneducated, or the badly-educated, to danger not actually at hand, that the number vaccinated has been trifling except when the small-pox was prevailing;" and "Still we have to regret that nothing but actual danger, or the belief in it, can induce the poor to seek the security of their families from a malady nearly as bad as the plague." Next we have the completion of Section 229.

"Improved Lodging-houses."

"The late Earl of Shaftesbury, in the debate on the Compulsory Vaccination Bill of 1853, observed that 'it was perfectly correct that the small-pox was chiefly confined to the lowest class of the population, and he believed that with improved lodging-houses the disease might be all but exterminated'."

The dissentients may agree with the Earl of Shaftesbury or they may not. If they agree, why do they propose the building of small-pox hospitals all over the country, and compulsory isolation of the infected, and recompense for compulsory quarantine of those exposed to infection, and burning of infected goods, and the retention of all the machinery of vaccine administration, with all the unavoidable risks of vaccination? What is the meaning of these recommendations if improved lodging-houses might all but exterminate the disease?

The next two sections are as follow:—

Small-Pox in Healthy Districts.

"230. In 1875 Dr. Farr constructed life tables based upon the vital statistics of, (1) All England, (2) Liverpool, and (3) healthy districts, in order to ascertain what effect healthy environment had upon zymotic diseases. His figures showed that—

"For every million born alive there would die, according to the life tables:—

	In Healthy Districts.	In England.	In Liverpool.
By small-pox ...	2,359 ...	6,521 ...	8,141
By fevers ...	28,146 ...	38,107 ...	76,563
By measles ...	6,912 ...	12,865 ...	26,973
By whooping-cough ...	10,234 ..	15,161 ...	34,021
By scarlatina ...	21,403 ...	30,021 ...	38,302

"Dr. Farr, commenting on these remarkable figures, states his opinion 'that healthy sanitary conditions as to food, drink, and cleanliness of person, house and city, stands first in importance; after it, but subordinately, come quarantine, vaccination, and other preventives, as means of subduing mortality; for the mere exclusion of one out of many diseases appears to be taken advantage of by those other diseases, just as the extirpation of one weed makes way for other kinds of weeds in a foul garden.'

"231. That the difference observed between the figures for Liverpool and the healthy districts is not merely due to the denser population of the former affording greater opportunity for infection, is, we think, shown by the fact that in industrial dwellings, where there is a considerable aggregation of persons upon area, but under superior sanitary supervision, there has been a marked immunity from small-pox. Dr. Southwood Smith long ago called attention to this fact (*Results of Sanitary Improvements*, p. 17); and we learn from the Secretary of the Improved Sanitary Dwellings Co. that in 1880-82, there were but two deaths from small-pox among more than 15,000 tenants, while there were 3,268 small-pox deaths in those years in London with a population of 3,800,000."*

What is a "Healthy District"?

In the above extract there lurks a very subtle fallacy. The reader is likely to assume that a "healthy district" is a district in which sanitation has been busy, and that, healthy districts in this sense of the term being grouped together, it is found that the mortality by small-pox,

* In the absence of data regarding the amount of vaccinal protection in the sanitary dwellings, statements about small-pox are valueless for the purpose in hand.

fevers, &c., is exceptionally low, and that this low mortality is a result of sanitary conditions which have been known to exist, and which have caused the district to be labelled "healthy." Now, as a matter of fact, on consulting Dr. Farr's Report it will be learned that the process of definition of a healthy district is exactly the opposite. In pp. viii. and ix., Dr. Farr writes:—

"Furthermore, including the London district of Hampstead, there are fifty-four large tracts of England and Wales which actually experience a mortality at the rate of only seventeen per 1,000—less by *five* than the average mortality per 1,000 of the whole country; less by *ten* than in nine districts; and less by *twenty-two* than the mortality reigning for ten years in Liverpool. Now the healthy districts have a salubrious soil, and supply the inhabitants with waters generally free from organic impurities. The people are by no means wealthy; the great mass of them are labourers and workpeople on low wages, whose families get few luxuries, and very rarely taste animal food. Their cottages are clean, but are sometimes crowded, and impurities abound; the sanitary shortcomings are palpable."

I have given this passage in order to show how easy it is to slip from the one conception of a healthy district to the other. Beginning with the fact that the districts have been purposely separated out from all the other districts in England because the statistics of the Registrar-General show that low rates of mortality have been experienced, there at once follows the statement about salubrious soil and pure water and clean cottages. Yet in the end the original idea of the healthy district is reverted to, for it is there admitted that the cottages are sometimes crowded, and impurities abound, and that "the sanitary shortcomings are palpable." Thus a "healthy district" may be a district with overcrowded houses, with abounding impurities, with palpable sanitary shortcomings. It may, in fact, be an insanitary district, using the term "insanitary" as referring to the absence of those means which human thought and skill have been able to apply to the prevention of disease. Notwithstanding the statement in Section 231, the one fact which, independently of a low death-rate, is common to the "healthy districts" (excluding Hampstead, Barnet, and King's Norton, as Dr. Farr does in his statistical calculations, on account of their propinquity to London and Birmingham), is that the density of population is low—that the average distance between the individuals residing on a square mile of ground is great. Thus, as Dr.

Farr points out (p. xxiii), in the healthy districts during 1861-70 there were 166 persons to a square mile; in all England there were 367; and in Liverpool 65,823, so that the mean proximity of person to person was in the three groups, 147, 99, and 7 yards respectively. In effect, therefore, a "healthy district" is one in which (*a*) the death-rate is below 17 per 1,000; and in which (*b*) however insanitary may be the surroundings of the dwelling-houses, however ill-ventilated and damp and dirty may be the houses themselves, yet the average distance of house from house, and hamlet from hamlet, and village from village, is such that ample fresh air is provided by nature, and that the opportunities of the spread of infection from one to the other are much less than in the rest of England, and especially than in Liverpool. The average distance of 147 yards between person and person which characterises the "healthy districts", can little more be claimed as a result of sanitary effort, than can the average distance which separates ships sailing on the Atlantic ocean. If, however, along with these advantages (*b*) of fresh air and low density of population, any rural district, owing to any circumstances whatever of unhealthy trades or otherwise, shows regarding (*a*), a rate of mortality exceeding 17 per 1,000 in the period embraced in the life table, then it is forthwith excluded from the definition "healthy district." The crux of the definition is that the mortality shall be low. What then does the Table given by the dissentients show? Not necessarily anything more than this: that in districts where the death-rate is low there is a low death-rate; that in districts where the death-rate is medium there is a medium death-rate; and that in Liverpool, where the death-rate is high, there is a high death-rate.

Age-Incidence in Healthy Districts.

It may be noted here that absence of opportunity for infection, owing to the high average distance between persons and houses and villages in a district, has the effect not only of diminishing the number of cases and deaths from infectious diseases as compared with districts where the population is denser, but also of lengthening the average time between birth and exposure to infection. The result is that in "healthy districts" individuals have more chance of escaping a disease like small-pox in their early years, and the share of small-pox and other infectious disease borne by children is diminished. Furthermore, in a healthy district, seeing that deaths are fewer and that, in particular, deaths in the early years of life are fewer, a

greater number of persons live on into adolescence and adult life. The result is that in any given ten thousand, or hundred thousand persons in a group of healthy districts, the proportion of the population at the higher ages will be greater than elsewhere, and the proportion of children will be less. This also has an important influence in making it appear that in a healthy district the age-incidence of an infectious disease is altered.*

Sections 232 and 233 refer to the Reports regarding Warrington and Dewsbury, which are not yet published.

The *Preussen*.

The small-pox outbreak on the steamship *Preussen* is discussed at length in Sections 234-5. In opening the subject, the dissentients say that in the evidence: "It was suggested that in such a case the influence of vaccination could be measured without the disturbing influence that sanitary circumstances are alleged to exert." Their own finding terminates with the statement that on the ship "Sanitation was conspicuous by its absence." The suggestion, therefore, is not overthrown but established. If all the data relating to the *Preussen* furnished by witnesses be summed up, so far as this is possible, the evidence remains in favour of vaccination. This is especially the case, if, with regard to the statement by the dissentients that: "There were also, in addition, the crew, numbering 120, who had been vaccinated and re-vaccinated; of these, fourteen were attacked and one died"; the further facts be noted that only six of the fourteen had been re-vaccinated previous to exposure, and that only four of the six had marks of re-vaccination. (Qs. 5,960-73).

Once more, in Sections 237-242, there is discussion of the influence of small-pox inoculation, and of the writings of Rast (this time without any marginal reference), Haygarth, Faust, etc.; but these have been considered already.

Sir James Simpson's Proposal.

It is with a sense of grave responsibility that I proceed to deal with the next Section in the dissentients' Statement. That Section is as follows:—

> "243. In 1868 attention was again recalled to the value of isolation in dealing with small-pox, by Sir James Simpson in a paper entitled *A Proposal to Stamp Out Small-Pox and other Contagious Diseases*, in which the success which had attended the stamping out system in dealing with certain animal pests was cited as an illustration

* See Section 197, *Royal Commission's* Report.

of what might be accomplished by an analogous system applied to the infectious diseases of mankind.

"The paper, which will be found at page 40 of the fourth volume of our Reports, is worthy of careful perusal. Sir J. Simpson's contention in brief was:—'For all that appears necessary for the purpose is simply the methodic temporary seclusion, segregation, or quarantine of those affected with small-pox, until they have completely passed through the disease and lost the power of infecting and injuring others. The pole-axe was the chief and leading measure required to stamp out rinderpest. *Isolation is the chief and leading measure required to stamp out small-pox.*' And he proceeded to show that by the Public Health (Scotland) Act of 1867, and less satisfactorily by the Sanitary Act of 1866 for England, the legislature had for the first time made such action possible to local authorities."

There is no doubt that careful heed should and must be given to the views of a medical man so distinguished in his own profession, and so well known to and highly thought of by the general public outside of the profession. It is quite natural, therefore, that in a document intended to influence the public and Parliament, his conclusions should be quoted and referred to. "The paper," say the dissentients, "will be found at page 40 of the fourth volume of our Reports", and it "is worthy of careful perusal."

Undoubtedly it is worthy of careful perusal, but it is not found there. What at a casual glance appears to be the paper is indeed there, but it is not the paper as published by Simpson. Here is how the matter is dealt with at page 40 of the Fourth Report. The evidence is that of Professor Crookshank:—

"Q. 10,984, Chairman: What is your next statement? —The stamping-out system as applied to human small-pox I should like to draw the attention of the Commission to this subject by reading the opinion that was expressed by Sir James Simpson. Sir James read a paper in 1868, and that was why I gave an account of the cattle plague and the meaning of the stamping-out system. I thought possibly some of the lay members of the Commission might not be acquainted with the term. Sir James Simpson

brought this same subject before the notice of the profession in 1868."

"Proposal to stamp out Small-pox and other contagious Diseases, by Sir J. Y. Simpson, Bart., M.D., D.C.L. Edinburgh: Edmonston and Douglas, 1868.

"The public mind has during the last two or three years become familiarised with the idea of stamping out a disease in the instance of rinderpest, a malady apparently spreading in this country, as small-pox does, by contagion only, and everyone knows the perfect success with which this affection has been lately banished out of England, while it has also, by due care, been prevented spreading to Ireland and the Isle of Man. I believe the same principle of stamping-out could be as successfully applied to the extirpation of small-pox among us as it has been applied to the extirpation of rinderpest, but of course with great differences.""

The quotation proceeds in the Minutes of Evidence from this point onwards in pages 40, 41, and 42 of the Fourth Volume of the Report. So begins the paper as given in Professor Crookshank's evidence. Here is how it begins as given by Simpson himself :—

"Proposal to stamp out Small-pox and other contagious Diseases, by Sir J. Y. Simpson, Bart., M.D., D.C.L. Edinburgh: Edmonston and Douglas, 1868.

"In despite of the marvellous protective influence of vaccination, the mortality produced by small-pox in Great Britain is still very great and startling. Like other contagious maladies, it varies much in the number of its victims from one year to another; yet, during the ten years from 1856 to 1865, small-pox destroyed in this island 51,034 individuals. In one of the last of these years, 1863, not less than 7,610 died from it; and in 1864 its amount of mortality reached 9,425."

After giving certain comparisons, and answering in the affirmative the question whether small-pox is preventable, Simpson proceeds thus regarding modes of propagation: "To understand the means to which I point, let it be premised that small-pox is—like scarlet fever, measles and whooping-cough—only a species of disease which, as a general rule, attacks once in a lifetime, and is only propagated from an infected individual to a susceptible individual by a specific poison generated in the course of the malady, and transmitted

from the affected to the healthy—first, by the near approach of one to the other; secondly, by their contact; thirdly, by direct inoculation; or, fourthly, by fomites, or by susceptible individuals being exposed to the virus when it has been imbibed into clothes, etc., with which the sick have been in contact. We would no more expect this known species of disease or poison to originate *de novo* at the present day, under any combination of circumstances, than we would expect known species of animal or plant—as a dog or a hawthorn—to spring up *de novo*, and without antecedent parentage."

Then he proceeds as follows, regarding vaccination and its place as a small-pox preventive: "The beneficial influence of Dr. Jenner's immortal discovery saves from death from small-pox, in our present population in Great Britain, probably about 80,000 lives yearly. As we have already stated, however, there still die from its ravages about 5,000 annually. Some among these 5,000 have been duly vaccinated, and yet are susceptible of small-pox after cow-pox, just as men formerly were found susceptible of a second attack of small-pox after they had passed through a previous attack of natural or inoculated small-pox. Others seem susceptible in consequence of the vaccination having been performed inadequately with imperfect matter, or without leaving vaccination scars of sufficient quality or quantity. Again, a large class of those that perish from small-pox consists of individuals who have never been vaccinated at all, or who happen to be exposed to the variolous poison antecedent to the age at which vaccination is usually performed. Doubtlessly a stricter enforcement of the new compulsory laws of vaccination, and a greater amount of attention to its proper performance with proper matter, will betimes diminish the number of the susceptible class. Yet, in the meantime, the disease still revels with fatal power among our population; and the question is, Can it be arrested in its progress"?

At length, after all these essentially important statements, Sir James Simpson proceeds: "The public mind has, during the last two or three years,"—and so forth, as given in the above extract from the Minutes of Evidence. At page 40 of the Report there is nothing to show that any such passages exist in the paper as I have quoted above, and in Professor Crookshank's evidence there is no hint regarding them. Let me note in particular the following statement from Simpson's

paper, as he himself gave it to the public: "The beneficial influence of Dr. Jenner's immortal discovery saves from death from small-pox, in our present population in Great Britain, probably about 80,000 lives yearly. As we have already stated, however, there still die from its ravages about 5,000 annually." What Simpson proposes is to endeavour to prevent, by means of isolation, the remnant of 5,000 deaths out of the 85,000 which he believes would, but for Jenner, have occurred annually. Indeed, as will be seen from the passages which have been quoted, he quite expected that under "the new compulsory laws of vaccination," the 5,000 would be still further reduced.

On page 42 of the Minutes of Evidence a hiatus is indicated in two places near the end of the paper. In the passage which follows the second hiatus, Sir James Simpson speaks thus: "My observations in the present communication refer especially to the stamping-out of small-pox, for I believe it is a malady whose extirpation could thus most easily be effected." Part of the omitted matter which precedes this quoted statement is as follows: "We cannot, as yet, by artificial prophylactic means, protect man against an attack of measles, scarlatina, and other contagious diseases, in the same way as we can protect him against the probabilities of an attack of small-pox by vaccination. We can only guard him by quarantine against the arrival of these maladies, and by isolation of the affected when once they do break out. Hence those maladies are, like small-pox in unprotected populations, often highly destructive when they do break out in localities or in communities which have not acquired any immunity by having previously passed through the same disease." It is on this account that Sir James Simpson believes that small-pox "is a malady whose extirpation could thus most easily be effected." In short, to quote the words of the Commission itself regarding isolation: "What it can accomplish as an auxiliary to vaccination is one thing; whether it can be relied on in its stead is another thing."

It does not lie with me to allocate responsibility for the manner in which Sir James Simpson's opinions have been put before Parliament and the public. As, however, my own paper is mainly in criticism of Dr. Collins and Mr. Picton, it is perhaps proper that I should point out that they may not have thought of consulting Sir James Simpson's paper in the original, but only as it appears in the Minutes of Evidence.

It may be proper also for me to state that, with regard to another question, No. 11,966 in Professor Crookshank's evidence, as given at page 101 of the fourth vol. of the Commission's Report, it fell to me in the course of my own evidence to point out to the Commission that, in a long quotation by Professor Crookshank from an old work—Dr. Beddoes' "Contributions to Medical Knowledge"—one sentence, and one only, had been unfortunately omitted from the body of the quotation, and that this omission had not been indicated by a hiatus, or by anything in Professor Crookshank's evidence; and that, from my point of view, the omitted sentence contained in it the whole explanation of Thornton's cases, which are dealt with in the quotation, and which otherwise would appear to tell against vaccination. In Section 44 of the dissentients' Statement, the subject matter of Q. 11,966 is mentioned, and a marginal reference is given to Q. 11,966. No note is made of the omitted sentence, and no marginal or other reference is given to Qs. 25,512-5, which point out the omission and its bearing on the rest of the quotation.*

Witnesses (and I was a witness), and possibly even Royal Commissioners, may, in spite of themselves, be more or less biassed on the subject of their evidence and deliberations, and I do not wish to say one word more than is necessary on this matter. But vaccination is, to my mind, a most important public health question; and the welfare, as regards small-pox, of future generations of the children of these realms may depend on Parliamentary action taken in consequence of the Report of the Commission, and the Statement of the dissentients, and the Minutes of Evidence. It is for this reason that I have thought it right to state these facts.

In Section 244, we read:

> "It has been largely in consequence of the experience derived from the great epidemic of 1870-72, in which the failure of the compulsory infantile vaccination system became so apparent, that attention has again of late years been directed to the necessity of providing proper hospitals for isolation, and to the enormous influence of such isolation in limiting outbreaks of the disease."

* See p. 107 ante. The omitted sentence is that in which Thornton writes: "I immediately procured some matter from a purulent pock which was the only one that was not degenerated into a sordid and painful ulcer."

In the same connection Dr. Seaton, Medical Officer of Health for Surrey, is quoted as to the evils of careless exposure of infected persons and things during the epidemic, and the too-late erection of hospitals under the influence of panic. About the need for hospital accommodation and disinfection there is little difference of opinion; the essential question here is whether hospitals can be trusted as a substitute for vaccination. I do not gather that Dr. Seaton is with the dissentients here.

London Small-pox.

In Sections 245-54 inclusive, London experience of small-pox from 1838 to 1895 is considered. The subject is introduced by a table of death-rates of small-pox, typhus, and scarlatina, so far as the data are available for these years. No figures are given for measles and whooping-cough, but regarding the table the remark is made:—

> "These figures confirm the conclusion to which the other evidence points, that while sanitary reforms have been followed by a reduction of the mortality from small-pox and fever, the recent development of proper hospital isolation has been most strikingly effectual in reducing almost to insignificance the mortality from those diseases in the case of which it has been most largely resorted to."

These questions have already been dealt with in the course of this review.

In Section 246 it is pointed out that hospital facilities and accommodation have improved for London, and it is added:

> "The effect of these increased facilities for treatment of small-pox has been that, while in 1871-2 only 31 per cent. of the small-pox deaths occurred in hospital, in 1893 87 per cent. took place therein."

But the small-pox deaths in London in 1871-2 were 9,657, and 31 per cent. of this was 2,994; while in 1893 the deaths were 206, and 87 per cent. of this was 179; so that the isolation of the 87 per cent. was a much less formidable undertaking than the isolation of the 31 per cent. Cause and effect have, perhaps, got into some confusion here in the minds of the dissentients. If the deaths had been only 50, the isolation might have reached 100 per cent.

In Section 247 the Report of the Hospitals Commission appointed in 1881 is quoted to show that London, from about 1860, had not shared properly in the dimunition of

small-pox which had been going on throughout the rest of the country for many years; and the Commission's recommendations are mentioned, these including compulsory notification and also, according to the dissentients, "removal of small-pox patients out of London, and the disuse of the intra-metropolitan hospitals for small-pox." Then it is shown that from 1882 to 1895 inclusive, the London small-pox deaths had been successively 430, 136, 1,236, 1,419, 24, 9, 9, 0, 4, 8, 41, 206, 89, and 55. In 1885, it is noted, cases began to be removed to the floating hospitals on the Thames at Long Reach, and in 1889 notification became compulsory.

What is the lesson derivable from these facts? A chief reason for the appointment of the Hospitals Commission was, that small-pox had not diminished in London as elsewhere; and that two or three observers, notably Mr. Power, of the Medical Department of the Local Government Board, had indicated that the very cause of this excessive prevalence was to be found in the existence in London of these hospitals for isolating and preventing small-pox. It was held that whilst vaccination was doing its beneficial work all over the country, as was being testified by diminished prevalence of the disease everywhere else than in London, the benefits of vaccination were being hidden in London by the establishment of hospitals which were centres of infection for surrounding districts. While small-pox had always been more prevalent in London than in the provinces, before 1871 its rate was seldom the double of theirs. But the Metropolitan Board Hospitals were opened in 1870-71, and after that a sixfold and sevenfold rate was reached. Notwithstanding the above quotation from the dissentients' Statement, the recommendations of the Commission did not give full effect to this evidence, but efforts were to be made to check hospital influence, by limiting the number of small-pox admissions, and by most careful and stringent precautions against intercourse with the surrounding zones of population. These efforts ended in failure. No amount of prohibition of personal intercourse succeeded in preventing the spread of the disease in the neighbourhood of the hospitals. The collecting together of a few cases in acute stages of the disease had the effect, under certain favouring conditions, and in despite of severe regulations against outside contact, of producing an infection which overleapt all bounds and carried the disease simultaneously to surrounding quarter-mile zones of population. Such extensions of the disease occurred inde-

pendently of the routes of traffic favoured by the hospital ambulances. When at length the lessons derivable from these facts came to have due heed paid to them by the Metropolitan Asylums Board, the disease was no longer treated in London hospitals, but ships for receiving and treating patients were established at Long Reach, on the Thames, at a sufficient distance from the metropolis. Since that time there has been little small-pox in London; and those who held that the effects of vaccination were being concealed by this counteracting cause have good ground for claiming that their contentions have been justified by the event. Lately, in London, the amount of vaccination default has become so great as to form a new factor in the case: a factor that is already showing its power in the increased share which children are bearing of such small-pox as has occurred within the last few years. The default, which was at a minimum in 1881, had become 10 per cent. in 1888, and 18 per cent. in 1892. It seems to me that the history of London small-pox for a generation back is strongly indicative of the dangers which would beset populations trusting to hospital isolation as a mainstay, and not merely as an auxiliary to vaccination.

Tramps.

In Section 254 reference is made to the influence of tramps and vagrants and common lodging-houses in spreading small-pox. I entirely agree that small-pox is often spread in this way, but the question is as to the remedy. In Dumbartonshire, in 1892, small-pox broke out in at least three separate centres in a navvy population of several hundreds occupied on a new railway. Navvies were coming and going from all parts of the country, and there was great risk of the spread of the disease. By means of thorough re-vaccination of the navvy population, along with isolation of infected cases, the disease was very quickly stamped out, and the navvies were at liberty to travel the country as they pleased without developing the disease and spreading the infection anywhere. To have attempted this without re-vaccination would have been, from my point of view, no less than criminal. So in Glasgow, when small-pox was last there, common lodging-houses were for a time almost transformed into vaccination stations.

Glasgow Small-pox.

Reference to Glasgow brings us to Sections 255-258, where that city is specially dealt with by the dissentients. The sections occupy two-and-a-half foolscap pages, and are too lengthy for quotation here. The statistics used are those of Dr. Russell; and I asked Dr. Russell if he would

Letter from Dr. Russell.

be good enough to comment on the use made of them. His reply is as follows :—

"Sanitary Chambers,
"Glasgow, 5th February, 1897.

"Dear Dr. McVail,—I rather resent the imposition upon me as a task the extrication of my authority from the line of argument followed by Messrs. Collins and Picton in their Grounds of Dissent from the Report of the Royal Commission on Vaccination. We all know what it is to write a full precognition of facts and opinions, and then in Court to be asked to testify to a careful selection of them. But that we recognise as the way of the special pleader. We do not expect such ways in the judge in his summing-up, yet Messrs. Collins and Picton think it consistent with their judicial function as Royal Commissioners to play the rôle of special pleaders. In 1895 I published a small book on *The Evolution of Public Health Administration in Glasgow*; in the latter half of which, having shown the enormous decrease *in the aggregate* of 'zymotic diseases,' I set myself (p. 63) 'to consider the chief infectious diseases individually, to bring out any peculiarity in the method of dealing with each, and to distinguish in the general bulk of success in prevention the special degree attaching to the treatment of each.' I pass each several disease under review, and point out how, while what may be called general sanitation has been effective more or less against the prevalence and fatality of all infectious diseases, and hospital isolation more or less against typhus, small-pox, scarlet fever, enteric fever, diphtheria, cholera, &c.; against each there have been special, one might even say specific, preventive measures taken. Thus, for example, against typhus, without the systematic suppression of overcrowding; against small-pox without systematic enforcement of vaccination, primary and secondary; against enteric fever and cholera without a pure water supply; against measles and whooping-cough without the control of schools, general sanitation, and even hospital isolation, disinfection and the ordinary means of prevention, would avail little. As to small-pox, I say (p. 69): 'Apart from their possibly sinister influence, hospitals can at best play only the part of an auxiliary in the campaign against small-pox. The first line of defence is primary vaccination. . . . (p. 70) Having secured as far as possible a primarily vaccinated population, the next line of defence against small-pox is still vaccination—vaccination reapplied as a system to every person of 10 years or upwards, and as a special process to all persons within the infected area which surrounds every case of small-pox.'

"I sum up (p. 73): 'In short, it may be said that the local authority of Glasgow has always extended to their medical adviser perfect freedom of action to do anything and everything to promote the prophylactic use of vaccine lymph.' Yet what do our special pleaders do? They announce their intention (p. 156) 'to review in some detail the history of small-pox and the various preventive measures which have at different times been in vogue, and to *scrutinise the grounds on which one alone of these preventive measures has been relied upon to the exclusion of others.*' Their method of doing this is to quote results which they well knew had been attained in Glasgow by the use of *all* 'these preventive measures,' *including vaccination and revaccination to an extent unparalleled in any other locality*; and they place these results to the credit of isolation and a vague something which they call 'sanitary reform'! Suppose another sect were to

arise of 'anti-gravitation-waterists.' This conception of honesty in the use of facts would sustain an argument that improved water supply had nothing to do with the suppression of cholera. It is all 'isolation' and 'sanitary reform' (limited)!!

"As I say, I resent being expected to waste time upon the exposition of every sophistical and dishonest use that may be made of my plain and unequivocal narrative of facts. No effort of mine will purge anti-vaccination literature of such abuses. The references to Glasgow experiences of whooping-cough and measles, and the implied argument therefrom may be burst up on the same lines; but I am not going to occupy myself any further with Messrs. Collins and Picton.

"Yours very truly,
(Signed) "JAS. B. RUSSELL."

Section 259 deals with Dr. Savill's Report on Warrington, but the Report is not yet published.

Section 262 says:—

"It is evident from the experience of Sheffield and Warrington that the most thorough carrying out of the vaccination laws will not prevent serious epidemics of small-pox, and that well-vaccinated towns cannot afford to neglect the provision of hospital isolation in order to prevent small-pox running riot in their midst."

Another reading of these experiences is that re-vaccination is required in supplement to primary vaccination, but they do not in the least support the proposal of the dissentients for removing all legal pressure with regard to primary vaccination.

Sections 263-272 are occupied with expressing the belief that the best way to prevent small-pox is by isolation and disinfection; but as the grounds for that belief have already been considered in detail, the belief must stand or fall with the foundation on which it is based.

"MEANS OTHER THAN VACCINATION."

Section 273 contains the following detailed list of means other than vaccination which should be used for protection of a community against small-pox."

"1. Prompt notification of any illness suspected to be small-pox. Improved instruction in the diagnosis of small-pox.

"2. A hospital, suitably isolated, of adequate accommodation, in permanent readiness, and capable of extension if required. No other disease to be treated at the same time in the same place.

"3. A vigilant sanitary staff ready to deal promptly

with first cases, and if necessary to make a house-to-house inspection. The medical officer of health to receive such remuneration as to render him independent of private practice.

"4. Prompt removal to hospital by special ambulance of all cases which cannot be properly isolated at home. Telephonic communication between Health Office and hospital.

"5. Destruction of infected clothing and bedding, and thorough disinfection of room or house immediately after removal of the patient.

"6. Daily observation (including, where possible, taking the temperature and inspection for rash) of all persons who have been in close contact with the patient during his illness; such supervision to be carried out either in quarantine stations (away from the hospital) or at their own homes.

"7. Closure of schools on the occasion of the occurrence of small-pox among the scholars or teachers.

"8. Hospital and quarantine stations to be comfortable and attractive, and so administered as to secure the confidence of the public. Hospital treatment to be free to all classes, and compensation to be paid to those detained or otherwise inconvenienced in the public interest, at the public expense.

"9. Tramps entering casual wards to be medically inspected, their clothing to be disinfected, and bath provided. The measures for detection and isolation of small-pox in common lodging-houses suggested in Section 507 of the Report to be carried out.

"10. International notification of the presence of small-pox, and special vigilance at sea-ports in communication with infected places, after the plan adopted in the case of cholera.

"11. Attention to general sanitation—prevention of overcrowding, abundant water supply, and frequent removal of refuse."

Prompt Notification.

Most of these recommendations belong to the alphabet of sanitation, and there can be no harm in their recitation in the Statement. But as to prompt notification, what about the numerous cases in which the disease is not diagnosed at the beginning, or at all? Here the individual who trusts to isolation as a protection is in an infinitely worse position than the individual fully pro-

tected by vaccination and re-vaccination, who needs to care nothing whether small-pox is diagnosed or not diagnosed, notified or not notified, isolated or not isolated. The latter can treat these things with indifference; to the former they may be a matter of life and death. Next, as to hospitals, what is "adequate accommodation" for an unvaccinated community?—for an unvaccinated London—for an unvaccinated England? Nobody knows; and in the interests of humanity it is to be hoped that nobody will ever have the opportunity of learning.

"Adequate" Hospital Accommodation.

Isolation by Compulsion.

"Prompt removal to hospital. . . of all cases which cannot be properly isolated at home" can mean nothing short of compulsion, as already foreshadowed in earlier parts of the Statement. Instead of the so-called compulsory enforcement of the "particular medical prescription" of vaccination, we are to have compulsory enforcement of the half medical and half non-medical prescription of Dr. Collins and Mr. Picton. That prescription, moreover, is not to be submitted to meetings of the democracy, as Dr. Collins at one time thought necessary with regard to notification and the mere provision of hospitals or hospital boards, when compulsory removal was not in question. Dr. Collins seems to have no time for such meetings now. Why? Is it because, following on vaccination default and neglect, he sees small-pox approaching at a pace that allows no time for the summoning of meetings? And then, what of the decisions of such meetings? Would he not have arrayed against him many of those who, in the school of anti-vaccination, have learned the art of rousing ill-will and passion against compulsion? What pictures might not be drawn of tender children torn from the arms of weeping mothers, and subjected to the un-tender mercies of hireling women in small-pox prisons? Is it not already being urged that medical men's errors regarding the diagnosis of small-pox make compulsory removal a tyranny to be resisted to the death? Do we not already hear that amateur and anti-vaccinating hydropaths can treat small-pox better in private houses than can medical men in hospitals? Then, as to actual resistance to compulsory isolation, have we not, even when the scheme is no more than adumbrated, the destruction of small-pox hospitals by infuriated mobs in the West of England, just as a foretaste of a probable outcome of the new compulsion? If Parliament is to settle the question of prevention of small-pox in accordance with extreme views as to the liberty of the subject, it will have

to reject unhesitatingly the dissentients' proposals for compulsory isolation. Members who require to consider how their votes in the House will affect the votes of their constituents can have no security that isolation will be less unpopular than vaccination.

Voluntary Isolation.

If then in securing the community against a drift back for a generation in the matter of small-pox to the conditions of a century ago, isolation is not to be insisted on any more than vaccination, what effect has this on the comparative value of vaccination and isolation? Let us suppose that both are to be voluntary, as the dissentients advise that vaccination should be. Under voluntary vaccination and re-vaccination, the individual is protected. He carries his protection with him wherever he goes. An adult has control over his own protection, and the father has control over the protection of himself and his child. Even under compulsory isolation there could be no complete security. The protection of the individual might fail at any moment. It would depend not on himself, but on other people. His *cordon* of protection would be a chain the measure of whose strength would be its feeblest link, and over not one link would he have efficient control. Failure of parents to observe the symptoms of illness; failure to call in a doctor; failure of the doctor to diagnose small-pox; failure in promptitude of removal; inadequacy of hospital accommodation; insufficiency of disinfection of persons and things—these would be among the risks to which even a law of compulsory isolation would leave him exposed. Obviously the risks of collapse of voluntary isolation would be much greater.

Voluntary Vaccination.

The proposals of the dissentients with regard to vaccination have next to be noted. Isolation, they state, is the better and more effective method of small-pox prevention. Vaccination, moreover, is positively dangerous to the individual. They "declare without hesitation" that "risk attaches to the operation of vaccination." As regards fatal vaccinations, they "give reasons for thinking the number of deaths under-estimated;" and they are "deeply impressed with the sad cases of severe illness and suffering and death" which result from vaccination. They are "struck with the fact that...in the houses of the poor...the operation cannot be regarded as free from even the more avoidable risks, except under conditions and precautions it is perfectly impossible to secure." Then, concerning erysipelas, they hold that "there is ample reason for believing" that

many cases have occurred which have never been certified as connected with vaccination; and they believe "that vaccination or the vaccine process is not infrequently proximately related to erysipelas, inflamed arms, ulceration, sloughing, and axillary abscess."

Their opinions concerning syphilis, and leprosy, and inflammatory and septic complications, and skin eruptions, and consumption, tubercle and scrofula, need not be detailed. In the face of all such dangers, and having at the same time found other measures which are safer, and more effectual, and more to be relied on than vaccination; and having, moreover, so utterly given up any views as to individual liberty that they advise that these other measures be made compulsory, there can be only one expectation as to what they must advise regarding vaccination. Clearly, it ought to be abolished root and branch, as a process not only needless but dangerous, and as a risk to which no parent can have any right to expose his child. What, then, do they recommend? Here is the Statement in Section 301:—

> "We therefore recommend that the law be amended by the repeal of the compulsory clauses of the Vaccination Acts. But in consideration of the prevalent belief in the value of vaccination as a prophylactic for an indefinite period, we suggest that in other respects the law should be left as it is, subject, however, to such modifications as are recommended for the diminution of attendant risks. The precedent established in the case of the abolition of compulsory church rates might be followed with advantage. In that case, all machinery for laying and collecting the rate was left intact, though the power of enforcement was taken away. The effect of our recommendation, if adopted, would be that vaccination would continue to be provided as at present for those who desire to avail themselves of it, but efforts to secure vaccination would be limited to moral influence—in a word, the whole country would be in the position of those unions in which the guardians have abandoned compulsion."

Instead, therefore, of vaccination being abolished, it is to remain optional. All its machinery is to be left intact, and efforts to secure it are not in the least to be prohibited, but are only to be limited to "moral influence." National

funds are to continue to be expended in giving parents the opportunity of exposing their children to risks (which, in the houses of the poor are declared to be unavoidable) of vaccinal disease and vaccinal death, at the same time that need for vaccination as a protection shall have been removed by means of compulsory isolation. The whole attitude is extraordinary, and an explanation, apart from "consideration of the prevalent belief," may well be sought for. It seems to me that no explanation sufficient to meet the case can be found excepting this: that the dissentients have more faith in vaccination and less faith in isolation than they are willing to admit, even to themselves. They see how in unvaccinated Gloucester small-pox broke out in a manner which foreshadows what may be looked for all over England if vaccination falls into abeyance. They want to be prepared for such an emergency. In addition to sanitation and "adequate" hospitals, the preparation is to consist in the maintenance of all the machinery for vaccination. Government is to be able to supply calf-lymph without stint. Officers are to be ready for carrying out general vaccination and re-vaccination. Funds raised by assessment are to be legally available to meet all expenditure. In short, instead of vaccination systematically carried out, there is to be panic vaccination; vaccination in the immediate presence of small-pox, and under circumstances in which the rigid and scrupulous care which the Commission recommends, and which the dissentients themselves so far homologate, will run risk of being neglected.

American Methods.

They own that they have been impressed with the remarkable freedom from small-pox in vaccinated children in recent epidemics. They have heard also, in the course of the evidence, that in some American States vaccination in the absence of small-pox is apt to be neglected; but that when small-pox has come to a community and has begun to claim its death-toll among the unprotected, then and there such vaccinal protection as can be given in the hurry and bustle of an epidemic is at once provided.

It is true that they do not in this connection mention American methods, but it is not easy to avoid thinking that, while they have hesitated to confess in set terms their faith (new-found or otherwise) in vaccination, they yet desire to give effect to it in their recommendations for the maintenance of everything vaccinal excepting compulsion. The new Anti-Vaccination League includes among its objects, "The Entire Repeal of the Vaccination Acts," and "The

Disestablishment and Disendowment of the Practice of Vaccination." The dissentients feel that they cannot go so far. In view probably of what they have learned during these seven years, the responsibility would be too serious; and no matter what their friends in the new Society may say or think, they cannot agree to deprive the country of the Jennerian prophylaxis. Their proposals, though ostensibly made as a concession to "prevalent belief," will work out, they may hope, so as to provide a protection not unlike that which Dr. Rauch described as existing in the United States; and though Dr. Rauch declared without hesitation that in his view the English system was much better, yet the dissentients are fain to content themselves with vaccination applied in presence of or under fear of small-pox, combined with isolation in hospitals which will be "adequate" or "inadequate", not so much in accordance with the number of beds in them, as in accordance with the amount of vaccination and re-vaccination in the community which they are intended to serve.

In the earlier days of vaccinal legislation in England, when Mr. Simon was a young man, he wrote his famous "Papers on Vaccination." These have received a permanent place in the literature of English public health. In concluding this review, may I quote to you a passage in which Sir John Simon expresses his opinion of vaccination, ripened and matured by a unique experience in the public health service of his country: "It is a favourite reflexion among philosophers that if departed great benefactors of our race could now and then look down on the harvest fields where mankind, age after age, is gladdened by the fruits of their labour, they would in general find themselves less remembered than perhaps their terrestrial ambitions had desired. Doubtless this is so; but let the noble compensation be noted, that often the thoroughness of a reformer's victory is that which most makes silence of the reformer's fame. For, how can men be adequately thankful for redemptions when they have no present easy standard, no contrast between yesterday and to-day, by which to measure the greatness of them? And to some readers that reflection may well occur at this present point, as they say their *Benedicite* for our workers of the eighteenth century. Of the present generation who in summer holidays enjoy their draught of cider in Devonshire, not many know that Baker unpoisoned it for them. Of those who go down to the sea in ships, not many have

Sir John Simon.

reading and imagination enough to contrast the sea life which now is with the sea life which was suffered in Anson's days, and to be grateful for Lind and Blane who made the difference. And in some cases ignorance best tells its tale by swaggering against the truce which protects it. At the anti-vaccination meetings, of which we now occasionally read, where some pragmatical quack pretends to be making mincemeat of Jenner, how small would become the voice of the orator, and how abruptly would the meeting dissolve itself, if but for a moment the leash were away with which Jenner's genius holds back the pestilence, and small-pox could start into form before the meeting, as our grandfathers saw it but a century ago."

Here we have an indication of the "moral influence" to which the nation would be handed over if the proposals of the dissentients were given effect to. The liberty of the subject would mean the liberty of imprudent neglect and delay; the liberty to despise a danger none the less real that for the time it is hidden; the liberty of a father to refrain from protecting his children against disease and disfigurement and death. And the "moral influence" in favour of vaccination would be small-pox unleashed, small-pox in its epidemic form, after isolation hospitals had been tried and found wanting.

At the conclusion of the discussion on Dr. McVail's paper, the following Resolution, submitted by the Council, in reference to the recent Report of the Royal Commission on Vaccination, was adopted by the Society:—

> "That this Society, while noting with satisfaction the pronouncement of the Royal Commission as to the value of Primary Vaccination, and the necessity for maintaining it, regret that, although the Report also brings forward conclusive evidence as to the value of Re-vaccination, the Commissioners have not seen their way to recommend legislation which would place it on the same basis as Primary Vaccination."

THE PREVENTION OF TUBERCULOSIS.

By JAMES NIVEN, M.A., M.B.

(*Read before the Epidemiological Society, March 19th,* 1897.)

It is now nearly thirty years since Villemin published his great work, *Études sur la Tuberculose,* in which he gives an account of his inoculation experiments: often briefly quoted in illustration of the fact that so long ago as 1865 positive evidence had been given of the communicability of this disease. This brief and passing recognition, however, scarcely suffices in acknowledgment of the philosophical boldness and masterly style with which Villemin unfolds the pathological problems of tuberculosis, the difficulties of heredity, the etiological factors and the affinities of the disease, especially with glanders; its distribution over different species of animals, and the experimental evidence not only of its inoculability but of its identity in man and in cattle. All the facts are marshalled in an orderly and complete fashion, and with what one is tempted to regard as an unerring clearness, round the central idea of infection. Villemin even goes so far as to surmise that the cause of the disease will be found to be of the nature of a germ, such as Pasteur had recently discovered, and to dream of a curative agent, such as Koch hoped tuberculin would prove. It would be altogether erroneous to imagine that Villemin's claim to be the author of the discovery that tuberculosis is an infectious disease rests entirely on his experimental work. His book is one sustained demonstration.

Some of the problems involved in the consideration of tuberculosis are doubtless imperfectly and briefly dealt with. But nothing is omitted, and the work makes, as a whole, an impression on the mind of the nature of a conversion.

Turning to Chapter XI, we find that he points out to us that tuberculosis is a malady of all climates: it is frequent in the tropics, and seems to diminish towards the poles. It is, thus, not the result of cold. It is rare or absent on elevated plateaux, in this respect resembling zymotic diseases generally.

It increases with aggregation of population. It is frequent in prisons, seminaries, barracks. It exacts a heavy tribute from the finest troops cooped up in ill-ventilated barracks, while it spares soldiers, even under circumstances of hardship, when on campaign. It is, thus, not due to physiological privation. These were indeed the circumstances which led Dr. Farr to regard tuberculosis as being probably caused by the inhalation of tuberculous matter.

It is rare or unknown amongst peoples living in the open air; it is frequent amongst individuals confined in houses. It is rare in malarial countries, because those are sparsely inhabited.

Amongst some peoples it was unknown until introduced by Europeans, but then spread rapidly. It is unknown amongst apes, in a state of nature, but very fatal to the same animals when confined and exposed to infection.

The poor man's cow fed by the side of the road is free from it; cows housed in stables—especially when they never leave the stable, as is liable to be the case in a large city—suffer heavily.

If now we turn to the experimental Chapter, Cap. XVI, we find an account of a considerable series of successful inoculations of tuberculosis in rabbits, and in one or two instances, in guinea pigs, by means of materials taken from tuberculous cadavers, as well as from tuberculous carcases.

Villemin does not neglect to control some of his experiments by leaving in the experimental stall rabbits not inoculated, all of which remained unaffected.

The identity of the disease in man and in the ox is inferred from the identity of the lesions produced in the rabbit. The track of the infection along the lymphatics is noted, as well as the multiplication of the infecting centres. Furthermore, the disease once induced in the rabbit is transferred to a second rabbit, and in one case to a third, by the introduction of tuberculous matter from the rabbit previously affected. It is manifest that, in this manner, all traces of the original matter introduced could be removed, and the disease thus shown to be a truly communicable one. He does not neglect to show in this way that the sputum of phthisical persons is infectious, and that some scrofulas also contain the tuberculous infection.

Thus, whatever may be said of individual experiments, the lines of experimentation were laid down, by which the whole subject could be elucidated.

To Villemin, in fact, belongs the double honour of placing the infectious nature of tuberculosis on a basis to

command the belief of thoughtful minds, and of expounding his discovery in a work which may still be read with delight and profit.

Amongst the distinguished men who succeeded to Villemin as workers in this field may be especially mentioned Chauveau, Cohnheim, and Salomonsen, though their work has been eclipsed by Koch's discovery of the tubercle bacillus. By his ingestion experiments Chauveau did not add to the proofs, already complete enough, which Villemin had afforded of the communicability of tuberculosis, but he removed the objection that the method of inoculation followed by Villemin was not that usually occurring in nature, and that it might be difficult to imitate nature.

What, then, is the value of Koch's addition to the knowledge of the disease?

In the first place, it helped to remove some refinements of doubt as regards the meaning of experimental inoculations. Was it not possible that, with the tuberculous matter introduced into the experimental animal, some catalytical principle was carried over which was the real cause of the disease? It is generally stated that this difficulty is put aside when, from the hundredth pure culture of the tubercle bacillus in succession, the disease can be reproduced unerringly.

It is difficult, however, to see wherein successive inoculation differs, in this regard, from successive cultivation. As Professor Delépine has pointed out, the essential element is multiplication of the ferment, which must therefore be organised. The inoculation experiments demonstrate this multiplication clearly.

But as regards the actual amount of the materies morbi in the different products of tuberculosis, the discovery of the bacillus gives the most valuable and even necessary light. It is in consequence of this that we have been led to recognise the immense importance of tuberculous sputum as an agent in infection: a fact which escaped the keen genius of Villemin, but on which Koch at once fixed.

Then, again, the actual demonstration of the tubercle bacillus has been of the greatest service in diagnosis, and in avoiding pathological errors; though it may be admitted that these difficulties may also be settled by the method of inoculation, if more slowly, yet with greater certainty.

The same slight differences in the results of inoculation with tubercular material from different animals, which Villemin observed, reproduce themselves in the cultures of bacilli from different animals.

For preventive medicine much has been gained by a study of the behaviour of the bacillus outside the body. Thus, we know definitely that direct sunlight rapidly destroys cultures of the bacillus (Koch, Delépine, Ransome).

We know by experiment that a considerable number of bacilli are necessary to cause infection by ingestion in the guinea pig, and we may be sure that this holds equally good for the lungs.

We are thus able to account for the great spread of the disease amongst people crowded in ill-ventilated and ill-lighted rooms. The limit of safety has been passed.

It is much to know that by means of air and light we can dilute and impair collections of infective matter into powerlessness for harm.

The question of increase of the tubercle bacillus outside the body at ordinary temperatures is an important one. In Koch's view, as given in 1882, this cannot well occur in such a manner as to be a serious factor in infection. But subsequent investigations have modified this standpoint somewhat.

It has been shown, according to Delépine, that the tubercle bacillus is capable of growing even on vegetable matter, kept in suitable conditions of moisture and not much exposed to sunlight. "I have watched," he says, "for months colonies of the human bacillus grow slowly in glycerinated media, at the temperature of an ordinary room, both summer and winter. At the end of that time the bacillus had lost none of its virulence, and was capable of giving typical tuberculosis to animals inoculated."*

He would infer that growth may take place in dirty rooms, especially if damp and dark.

From these illustrations it is clear that a precision and fineness is thus added to our knowledge of many obscure points in the natural history of tuberculosis, which we could not have gained without the discovery of the bacillus.

Then, again, the discovery of tuberculin, which is a direct outcome of the isolation of the bacillus tuberculosis, is of the utmost importance for the diagnosis of tuberculosis in cattle, and probably ought to be much more freely used for that purpose in man.

All this, besides a much more intimate knowledge of the disease, we owe to Koch's discovery.

* *Journal of State Medicine*, March 1896, page 82.

It should be mentioned that Koch repeated on a large number of guinea pigs the inoculation, ingestion, and inhalation experiments of his predecessors with the pathological products of tuberculosis as well as with suspensions of pure culture; and that in this way also he proved most conclusively the infectious nature of these materials by the different channels mentioned, as well as the identity of the disease in a number of warm-blooded animals.

We cannot do more than mention a few of the points which have been elucidated by Koch's beautiful discovery, and by his demonstration of the *rôle* played by the tubercle bacillus. But what is of more consequence than all the isolated advantages which might be enumerated, is the rapid effect which these researches had in leading a vast number of energetic workers into the same field, and in popularising a knowledge of the infectious nature of the disease: so that it is now the exception to find any well-informed man who disputes its communicability.

For the purpose of our theme, however, one or two points are of cardinal importance.

In the first place the infective potency of sputum, when it has been reduced to dust, was clearly enunciated by Koch in his first communication, as well as the intimate part which it must necessarily play in the propagation of tuberculosis. The exceptional power which sputum possesses depends on the immense numbers of bacilli discharged in it, especially during the advancing and active stages of the disease.

In the same manner we know that tuberculous ulceration of the bowel is attended with a copious development of bacilli, which must be discharged in the fæces, and constitute a serious source of infection, the more so that abdominal tuberculosis is often not recognised for a time.

The dried fæces will certainly be highly infectious, and will therefore form an important source of infection, especially in children suffering from tubercular diarrhœa.

Leaving now the question of the discovery of the tubercle bacilli and of its etiological relation to tuberculosis, and fixing our attention on the sputum, which was recognised by Koch as the chief agent in conveying infection, and in which, as Schill and Fischer demonstrated, infective virulence might be retained for as long as 160 days, it is obvious that the question would forthwith arise: If the sputum is capable, when dried and trodden into dust, of retaining and disseminating infection, whether some proof of the infec-

tivity of such dust could not be obtained. This question Cornet set himself to solve. The results of his work are recorded in his book *Über Tuberkulose*, published in 1888, of which a summary is given by Dr. G. A. Heron in his work on the *Communicability of Consumption.*

It is impossible here to do more than indicate, in outline, Cornet's method of experimentation. Selecting the dust of rooms and wards occupied by consumptives from situations which were not directly accessible to sputum derived from the phthisical inmates, he removed it from about one square metre of surface, mixed it with a suitable sterilised medium, and injected it into guinea pigs, of which generally three were used for each experiment. Needless to say, the work was carried out with all necessary precautions against accidental contamination.

A certain proportion—sometimes all the experimental guinea pigs—died from the effects of other infective matters in the dust. Nevertheless, the majority survived, and it was possible, owing to the large number of experiments made, to arrive at certain conclusions.

These may, for our purpose, be briefly stated as follows:—

Seven hospitals, containing 21 wards with phthisical patients, were examined by insertion of the dust into 94 guinea pigs; of these 52 died of other diseases, and in 15 instances at least one guinea pig became tuberculous, representing 15 wards in 5 hospitals. The wards of three lunatic asylums were examined, experiments being made on 33 animals, of which 16 died of other diseases than tuberculosis. In three instances, one guinea pig became tuberculous. Two prisons gave negative results, as did a chamber used for inhalation experiments with tuberculous materials. From the rooms of tuberculous patients treated at home, tests were applied in 62 instances to 170 guinea pigs, of which 91 died of other diseases, while in 21 instances at least one guinea pig became tuberculous. In 22 instances the animals remained sound. The injected material failed to produce tuberculosis when taken from a policlinic, from near a lupus patient, from an orphanage, from the Pathological Institution, from surgical wards, and from streets.

The most important point noted was that, where the sputum had been carefully collected or destroyed, and not allowed to contaminate the room, there the inoculated dust failed to produce tuberculosis. These experiments conclusively do away with the idea that the infection of

tuberculosis is everywhere present in amount sufficient to set up the disease.*

It should be added that repeated examinations of the dust, microscopically, failed to reveal any bacilli, so that the infective matter in such dried sputum exists chiefly as spores.

These highly important experiments, therefore, make it clear to us that the infective power of matter derived from consumptives is strictly local, and is only present when perfectly possible precautions are neglected.

It is not to be discovered, even by so sensitive a test as inoculation of the guinea pig, in the dust of streets : a fact, surely, of cardinal importance. Still further to elucidate this point, Dr. Cornet made an inquiry into the incidence of phthisis on the street-sweepers of Berlin, and found that it was surprisingly slight.

There can be no doubt also, that, in the absence of sufficient care with regard to the discharges of the sick, their surroundings are a source of danger to those inhabiting the same rooms.

The link would be complete if we could trace the actual occurrence of infection from the infector to the infectee. Numerous isolated instances of apparent infection have been recorded, and a useful collection of such instances may be found in Dr. G. A. Heron's book. But isolated instances are insufficient. The history of the subject enables us to say, with confidence, that the infection must have been derived from some source; but it is manifest that the multiplicity of the sources tends to throw doubt on any one etiological factor, especially where the cases are isolated.

This is less true of tuberculosis the result of accidental inoculation of tuberculous matter, and Senn's work on *Surgical Tuberculosis* may be usefully consulted for instances of this nature.

No possible doubt, however, could exist as to the source of the infection which swept away the inmates of the Catholic nursing institutions of Prussia into which Dr. Cornet instituted his exhaustive inquiry.

These institutions were recruited by selected lives—viz., women of more than ordinary physique, at the most vigorous period of their existence, and bearing no relation to each other. Yet within two or three years of their

* See also "Experiments on the Relation of Dust in Hospitals to Tuberculous Infection", by G. A. Heron, M.D., and T. H. Arnold, Chaplain, B.A., M.D., Cantab.—*Lancet*, Jan. 6th, 1894.

entrance into these institutions, the number of deaths from phthisis was terrible. No doubt the condition of the institutions was insanitary, and the work excessively hard and risky for the novitiates. But there is equally little doubt that the enormous death-roll recorded in Cornet's paper was due to the infection from previous victims—infection to which he shows the novitiates to have been specially exposed.

An excellent account of this inquiry is given in Dr. Heron's book. Those who wish a fuller account may refer to Cornet's work, *Über Tuberkulose.*

No account of any pestilence with which one is acquainted bears, on the face of it, a plainer history of infection.

Anyone who may choose to take the trouble may trace for himself a similar story in the houses of consumptives. In the course of a second inquiry which I made in 1890 into a hundred deaths in Oldham, certified to be from tuberculosis—selected, it is true, as far as possible from people above the lowest stratum of society—I found that, in about one-half of the cases, a history could be obtained of prolonged and intimate exposure, on the part of the deceased, to infection.

In a recent inquiry, which I have had made by Sanitary Inspectors, into the uncertain class of deaths described as tabes mesenterica, the same history of exposure to infection comes out, though to a slighter extent.

It might be objected that, in many of these presumed cases of infection the infector and the infectee belong to the same family, and that the element of heredity thus comes in. As we shall see, however, there is reason for believing that the results are but slightly diminished in value on that account.

Considering the length of period between the reception of infection and the development of tuberculosis, and the oblivion into which exposure to infection often shortly falls, it is surprising in how many cases prolonged and intimate exposure can be traced. This is the case simply because it is often necessary for infection that exposure should be intimate and prolonged, so that the infecting bacilli may have the opportunity of evading the numerous difficulties which they have to encounter before they can reach the lungs, at the same time that they catch the organism off its guard.

It is, perhaps, needful to say something of the immunity from phthisis which the nurses at Brompton Hospital have enjoyed, and which has been so often alluded to. The

reason of this probably lies in the circumstance that a certain amount of infective matter, a certain number of bacilli, are necessary to produce infection. The wards of Brompton Hospital are well lighted, well ventilated and clean, and the work of the nurses is probably in no way comparable to that of the poor novitiates of the Prussian Catholic institutions.

The nurses at Brompton are, no doubt, under healthier circumstances altogether than the Catholic nurses; while, as regards their work and opportunities of exposure, the novitiates would represent nurses and ward-maids together.

The circumstances of a poor home and of many lodging-houses, have probably much closer affinities with the Catholic nursing institutions than with Brompton Hospital.

In fact, the infection of tuberculosis only displays itself in that virulence of which it is capable when it has attained a certain degree of concentration.

The story of the Catholic nursing institutions is so striking and so complete, that it would be perhaps well to dwell upon it a little longer. I shall, however, not go into the varied statistics by which Cornet elucidates the nature of the occurrence, and which are given by Dr. Heron, but content myself here with quoting briefly from his account of the research:—

"The members of these Orders are bound, by a vow, to remain for life in their respective Orders. As a consequence, their life-histories have been obtained with almost perfect accuracy. They relate to the twenty-five years preceding 1888. Thirty-eight convents were selected, because their reports furnished the most reliable and fullest information. These communities represented a yearly average of 4,028 persons; and, as Cornet observes, a sum total of 87,450 years of human life passed under observation, with 2,099 deaths.

"The death-rate of the general public from tuberculosis is from one-seventh to one-fifth of all deaths. Instead of that mortality, we find amongst these nursing institutions that nearly two-thirds of the deaths, or 62·88 per cent., are due to tubercle alone.

"Cornet states that these convent communities are composed of persons whose health at first is known to be excellent, admission being dependent on the production of a medical certificate to that effect.

"After the first year, the number of deaths from tuberculosis amongst persons entering the convent rapidly

attained a maximum, and continued high for the next few years."

In an inquiry into deaths from tuberculosis in 1886, recorded in the report on the health of Oldham for that year, I mentioned that the most common period between infection and death appeared to be between two and three years; which agrees with Cornet's striking figures given in 1888, and seems to a certain extent to point to infection being the cause of death in both cases. To resume :—

"In the first five and a-half years of convent life, out of 709 deaths, 494 are due to tubercle alone. In the nurse's third year of cloister life, tuberculosis reaches its deadliest period. A healthy person of twenty-five years of age, who then begins cloister life and the work of a nurse, has an expectation of life equal to that of a healthy woman of the general population, who is fifty-eight years of age."

This is, however, by no means an isolated history. The excessive death-rate from phthisis, which formerly prevailed among the home troops crowded together in barracks, and the rapid spread of phthisis in seminaries, prisons and penitentiaries, point in the same direction.

The Report of the Royal Commission on the Organisation of Military Hospitals, &c., issued in 1858, gives on page xv the following table for the year 1853 :—

Rates of Deaths per 1,000 of Mean Strength by	Household Cavalry.	Dragoon Guards and Dragoons	Foot Guards.	Infantry of the Line.	Civil Population of 24 Large Towns.
Inflammation of the lungs, pleurisy, and acute catarrh	0.2	0.7	1.3	1.3	0.5
Spitting of blood, consumption, chronic catarrh, asthma, aud difficulty of breathing	6.4	6.6	12.5	8.9	5.8
All causes	11.1	13.6	20.4	17.9	11.9

And goes on to state that, as other possible influences may be excluded, these fatal results may be ascribed to the crowded and insanitary condition of the barracks. The difference in mortalities is ascribed to differences in the barracks, and facts are adduced to prove that in the absence of such conditions, this fatality does not occur. The subsequent great reduction in the mortality from phthisis under better conditions amply justifies the conclusion drawn.*

* "The Prevention of Consumption", by J. Edward Squire, M.D.—*Transactions* of the Epidemiological Society, N. S., vol. viii.

In order to illustrate the effect of crowding people, many of them phthisical, into an insufficient space, especially as regards day room, I have obtained the death-rate in a certain number of common lodging-houses in Manchester, for comparison with that of the city generally: premising that although the disease, so far as individuals are concerned, may not have been contracted in these particular lodging-houses, we may assume that the general conditions existing throughout the country are not worse.

Particulars have been ascertained in regard to seventy-seven Manchester common lodging-houses, containing an average total of 1,825 persons. During the years 1893-94-95, there were removed to Crumpsall Workhouse from these lodging-houses 108 persons who died there of "phthisis"—two others made the number up to 110—giving an annual death-rate of slightly over 20 per 1,000. Referring to the Report on the Health of Manchester for 1895, we find that the highest mortality from tubercular diseases at any age group, viz., at ages 25-45, was 3.95. This will give an idea of the amount of infection which is being constantly produced in these insanitary houses.

Evidence to the same effect is given by Dr. Ransome in his paper on tubercular infective areas.

I have dwelt at some length on clinical investigation, because it is open to everyone to undertake this line of inquiry, and because it is desirable that fresh investigations should be undertaken. We may now inquire what aid we can obtain from statistics.

On some Statistics relating to Phthisis and other forms of Tuberculosis.

Phthisis is by no means, generally, one of the most difficult diseases to diagnose, especially in the adult. Moreover, over large masses of figures we are at first inclined to suppose that the same errors are liable to occur—at all events for a limited number of years.

Hence, when we find that there has been a marked though gradual diminution in the death-rate of England from phthisis, from 2.41 per 1,000 in 1870 to 1.39 in 1894, we are bound to admit that, probably, the larger part of this decrease is real. Doubtless some of it is due to greater precision of diagnosis, and I therefore put side by side with it the gradually increasing-death rate from cancer.

To what extent this reduction is a real one may, in some

degree, be determined by taking the death-rate from other forms of tuberculosis.

It is certain that other forms of tuberculosis are, in part, conditioned by the amount of phthisis prevalent, although the relation is not a constant or close one. This, however, may be as much the fault of defective diagnosis as of facts.

If we run our eye up the figures (with the exception of 1894, which was in all respects an exceptional year), we do not find the same reduction in the death-rate from other forms of tubercular disease, although the birth-rate has been a declining one.

This fact must give us pause, I think, and induce us to look round for some explanation. We have an increasing aggregation of people in towns on the one hand, and an increasing provision of sanitary dwellings, with sufficient air space, and breathing room around them, on the other. Is it possible that the deaths assigned to other forms of tuberculosis are due so largely to other forms of infection—for example, to the consumption of tuberculous milk—that no parallelism is to be expected? We cannot think so, while allowing to this consideration its full weight.

Professor Delépine, in his paper in the *Journal of State Medicine*, March 1896, states, however, that there is some reason for supposing that tuberculosis amongst animals is increasing.

We must assume, I think, that the improvement inferred from the phthisis death-rate is not so great as it looks.

Let us take as a basis of reasoning the table of mortalities from phthisis given in Newsholme's *Vital Statistics* for different groups of ages, extended up to the year 1894.

Mortality from Phthisis in England and Wales per million living at successive Age Groups.

Years.		All Ages	0 —	5 —	10 —	15 —	20 —	25 —	35 —	45 —	55 —	65 —	75 —
1848-80	Males	2443	1077	448	628	2093	3687	3941	4089	3857	3317	2152	752
	Females	2413	1033	503	1077	3019	3809	4175	3842	2830	2124	1364	546
1851-60	Persons	2679	1305	572	1025	2961	4181	4317	4091	3466	2840	1983	808
1861-70	„	2475	968	454	825	2651	3928	4243	4026	3340	2656	1603	539
1871-80	„	2116	767	358	664	2036	3117	3619	3745	3132	2449	1476	492
1881-90	„	1724	536	290	521	1545	2324	2901	3132	2737	2169	1355	521
(5 years) 1890-95	„	1524	473	248	433	1330	1929	2436	2876	2521	2034	1205	472

The first thing which strikes us about this table is the enormous reduction in the number of deaths from phthisis at early ages.

The reduction is at ages 0—5 from 1,305 in 1851-60 to 536 in 1881-90, with corresponding figures for other of the lower ages. We cannot hesitate in affirming, especially having regard to the great advances made in medical knowledge, that great part—possibly the greater part—of this reduction must be put down to improved diagnosis. If, however, this be so, then the same will hold, to some extent, in regard to more advanced ages; and we are left in doubt as to whether a considerable part of the improvement in the phthisis death-rate is real or only apparent. Our doubts are still further increased when we find that the number of deaths ascribed to phthisis at ages 0—5 is steadily much in excess of the number at ages 5—10. The death-rates from phthisis recorded in the registers for 1881-90, per 1,000,000 of the population, are as follows:—

Under 1 Year.	1 —.	2 —.	3 —.	4 —.
961	802	379	262	243

This is quite incompatible with Boltz's figures given further on. I do not wish, however, to dispute that there has, probably, been a marked reduction of the phthisis death-rate at the earlier ages, but only to point out the need for amended diagnosis. The increased care bestowed on children, and a long experience of beneficent factory legislation, not to mention improved ideas of prophylaxis must, assuredly, have made no inconsiderable change.

Compare with Boltz's statistics Siedamgrotzky's figures of animals slaughtered in 1889 *in the principal abattoirs in Saxony.—Strauss, p.* 540.*

	Of	Tuberculosis.	Percentage.
123,725	Calves from 0 to 6 weeks ...	8 ...	0.006
797	Bullocks and heifers from 6 weeks to 1 year	3 ...	0.3
6,283	Oxen and cows from 1 to 3 years...	444 ...	7.0
14,404	„ „ from 3 to 6 years...	1,346 ...	9.3
12,110	„ „ over 6 years ...	1,943 ...	16.0

Still, I think, we may assume that the figures for adult life are not so far wrong, and at all events we may rely on the diagnosis sufficiently to give value to the figures for the army, navy, prisons, and various institutions.

Then, again, we are, I think, in still greater doubt when we come to appraise the diagnosis of tuberculosis in its more obscure forms. When we add to the doubtfulness and variability of the diagnosis the varied circumstances of

* It is well, at the same time, to remember that a certain amount of disease in young animals does not figure in the experience of abattoirs.

age constitution of the population, we are soon involved in grave doubts as to the value of our statistical inferences.

This, then, I call the inferior order of statistics. It is a blunt weapon, which can only be confidently used for rough purposes. With this understanding I subjoin figures from a paper by Mr. Welton relating to phthisis, read before the Manchester Statistical Society January 1897, showing the mortality from phthisis per 1,000 living for the years 1881-90, at ages 10 to 65, in London and some of our agricultural districts.

Mortality from Phthisis per 1,000 *living in London and some Agricultural Districts,* 1881-90 (*extract from Mr. Welton's Paper*).

Districts.	10—15		15—20		20—25		25—35		35—45		45—55		55—65	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
London	0.29	0.53	1.17	1.20	2.26	1.62	3.80	2.59	5.30	1.24	5.47	2.58	4.64	1.84
Kent ...	0.31	0.65	0.98	1.73	1.94	2.12	2.62	2.50	2.97	2.48	2.88	1.99	2.31	1.45
Essex ...	0.31	0.67	1.00	1.63	1.78	2.03	2.36	2.21	2.87	2.53	2.90	1.98	2.64	1.48
Hampshire ...	0.22	0.56	1.27	1.78	3.03	2.34	4.12	2.93	4.20	2.88	3.66	2.12	2.57	1.47
North Wales	0.44	1.12	1.80	3.21	3.44	3.69	3.68	4.00	3.22	3.21	2 84	2.21	2.99	1.90
Lincolnshire	0.30	0.86	1.18	2.29	2.20	3.03	2.45	3.31	2.36	2.38	1.98	1.56	1.76	1.14
Hereford shire	0.21	0.55	1.11	1.64	2.09	2.52	2.29	2.58	2.20	2.21	1.91	1.25	1.57	0.98
Monmouth shire	0.30	0 57	0.98	2.12	1.65	2.55	2.19	3.15	2.19	2.60	1.84	1.68	2.04	1.14

On examining these figures, the first thing which strikes us is the predominance of phthisis in the rural districts at the younger ages. Why is this? Is it owing to the survival of a defective nomenclature, or is it because the more sturdy part of the rural population migrate into our large towns? But such migration will not take place before the age of 15—nor to a great extent before the age of 20—and it is precisely at the ages 10-20 that the predominance in phthisis of the rural districts is exhibited.

Is it, perchance, owing to the defective habitations of the rural populations, or to infection from cattle? But such infection would be least liable to occur at so early an age, unless by means of milk.

These inequalities require investigation.

If it is that diagnosis is defective (and, from the very

meagre occurrences of second cases in a house, which I obtained from a search and tabulation of the Oldham death-records for eleven years, as compared with the abundant evidence of multiple occurrences yielded by clinical inquiry, I am inclined to think that we have still much defective diagnosis to contend with), then a serious barrier is thereby opposed to preventive measures.

Until the public have become aware that a chronic bronchitis is not unlikely to be in reality phthisis, and not merely to pass into it, we shall be much hindered; but we shall be still more impeded if deaths are classed as bronchitis which ought to be recorded as tuberculosis.

It is true, the distinction is by no means always an easy one. But the examination of sputum is now made extremely simple, and there is no longer a sufficient reason why the sputum of every case of continued bronchitis should not be repeatedly examined for tubercle bacilli.

Moreover, we have a much easier method of diagnosis by the injection of tuberculin. There is no doubt of the dangers attending the use of tuberculin, when used in large amounts and injected day after day. This is well shown by the facts collected by Professor Delépine.

Whether, however, used in a dose only sufficient for diagnosis, its effect might not be at once both diagnostic and curative, is well worthy of inquiry. I cannot think that such a use of tuberculin is forbidden by any facts which we at present possess.

Where there is the slightest reason to suspect tuberculosis the nature of the disease should be cleared up in the interests of others than the patient, either by examination of the discharges or by the use of tuberculin: where neither method is applicable, as in cases of tubercular meningitis, and in young children generally, by post-mortem examination.

One class of case especially is, I believe, often a source of serious danger, viz., that of a child suffering from tuberculosis of the bowel. The fæces are discharged and dried on the child, with the result that infection is dispersed, as in typhoid fever. The child appears on the death-certificate as having died, perhaps, of summer diarrhœa, which no doubt has been the immediate cause of death. Many adults, moreover, suffer, I believe, from tubercular diarrhœa, with the same consequences to the immediate attendant as result from diarrhœa in typhoid fever.

By one or other of the means mentioned the true nature of such cases ought to be elucidated.

Before leaving the subject of the inferior order of statistics, I may mention Longstaff's oft-quoted calculation in reference to the transmission of consumption between man and wife. I need scarcely say that, beautiful as is the mathematical part of the statistical reasoning, the result is useless, inasmuch as no reliance can be placed on the extent to which the experience of transmission which he uses expresses the general fact.

Statistics really valuable as guides to action are those founded on exact pathological observation.

As an example of such useful statistics, I quote the following from Dr. Ransome's *Milroy Lectures*, p. 32:—

"My colleague, Dr. Harris, has himself investigated this subject, and found that, in at least 39 per cent. of the autopsies at the Manchester Royal Infirmary, there was evidence of cured phthisis. He also refers (in a paper on the curability of phthisis) to the researches on this point of Cruveilhier, Rogée and Dejerini, 50 per cent. of their cases having shown signs of caseous and calcareous formations. Bollinger, in 256 similar cases, found 27 per cent. with evidence of healing, Standacker, 20 per cent., and Marsini 31 per cent. Evidently a very large proportion of our population suffers from tubercular disease, and recovers from it."

A hopefulness and just prospect of recovery may thus be presented to tuberculous persons, while the incitement afforded to early diagnosis should be of immense value to preventive medicine. Another point strongly suggested by these figures is the great amount of maiming which is going on, short of complete disablement.

As another illustration of the value of statistics, founded on exact observation, may be given the statistics of pathological records of tuberculosis at an early age. If the statistics of tabes mesenterica are truly represented by the death-certificates regularly sent in, we must inevitably conclude that a vast number of children are born with congenital tuberculosis.

How slight in reality is the foundation for such an idea is conclusively shown by the following quotation from a paper by Professor Delépine on tuberculous infection through the alimentary canal.

"The following statistics are given by Boltz in his *Inaugural Dissertation.* Out of 2,576 children whose bodies were submitted to a post mortem examination in Kiel between 1873 and 1889, there were 424 cases of tuberculosis, or 16.4 per cent. The following table shows the percentage at different ages of the tuberculous deaths:—

"Still-born children	...	...	0.0 per cent.
Up to 4 weeks old	...	...	0.0 per cent.
5 to 10 weeks old	...	...	0.9 per cent.
3 to 5 months ...	...	...	8.6 per cent.
6 to 12 months ...	...	...	18.3 per cent.
1 to 2 years ...	...	...	26.8 per cent.
2 to 3 years ...	...	...	33.0 per cent.
3 to 4 years ...	...	...	29.6 per cent.
4 to 5 years ...	...	...	31.8 per cent.
5 to 10 years ...	...	...	34.3 per cent.
10 to 15 years ...	...	...	30.1 per cent."

Further statistics to the same effect are given in Strauss's treatise, *Sur la Tuberculose et son bacille*, in the Chapter on Heredity.

Our large abattoirs might be made to yield statistics most important for human etiology.

Similarly exact pathological records at children's hospitals might be made available for similar conclusions. As it is, the post-mortem observations of Sims Woodhead on children are of much value in this connection.

On the Modes of Communication of the Disease.

Turning now from the value of exact statistics and the possibility of obtaining them and utilising all the information which we possess, let us consider some of the modes by which infection is likely to be communicated; and, first, let us consider the modes of communication from one human being to another.

Unquestionably, the greatest source of infection is dried sputum, whether this is dried on the floor, in a pocket-handkerchief, on the bed-clothes, or on the person of the feeble and neglected consumptive. In the early stages of the disease it is difficult to say which is the more dangerous to others: sputum discharges on the floor, or in the pocket-handkerchief.

For reasons already given, I am disposed to think that tubercular excreta are a decided source of danger.

Then, again, the copious discharges of tubercular ulcers and of tubercular bones cannot be regarded as free from danger, especially when we remember that pus, which reveals no tubercle bacilli to the microscope, may convey the disease to the guinea pig, when inoculated. All tuberculous discharges must be regarded as dangerous when in the dry condition.

These are obvious facts, and the prophylaxis is easy.

There are, however, other sources of infection, many of them such as very easily to escape attention.

Thus, for example, all articles manufactured by consumptive tradespeople are fraught with danger more or less to those who use them, according to the stage of the disease, and the opportunity which the manufactured article has of accumulating infection. Particularly is this the case in regard to the manufacture of articles of hair, or of any material which gives off sharp or loose fragments, and which is not subsequently freely exposed to light and air. On account of the massiveness of infection, woollen and cotton waste must thus occasionally be the source of infection.

It is probable that the disease is thus not rarely conveyed in garments made by consumptive tailors, milliners, sempstresses, etc. Cotton and woollen waste are largely used for stuffing furniture and bed-mattresses, practically undisinfected, and in this shape must occasion mischief to some extent.

The same is true of rag flock.

Yet in no instances are workpeople obliged to take the necessary precautions, which are easy in the extreme.

Then, again, instruments used in clinical work are liable to convey infection, unless carefully disinfected after each use, as Cornet has abundantly shown. Particularly is this applicable to the use of the spatula, and even more particularly to the employment of the throat mirror and other implements used in diseases of the throat. It is to be feared that the disease may sometimes be conveyed in this way, especially as the bacilli are abundant in ulcers of the mouth.

Children have many opportunities of contracting infection in consumptive households.

They are often allowed to crawl about the floor, and thus to inhale the infection which is specially abundant in that situation. Or perhaps they put their soiled little hands into their mouths. If the mother is consumptive, infection is apt to get on her hands from a soiled handkerchief. She takes the hands of her infant in hers, and the child forthwith thrusts its thumb into its mouth.

A consumptive mother must be instructed to the utmost in the precautions necessary to avoid infecting her child.

Generally speaking, of course, there is danger of abdominal tuberculosis being acquired by the subsequent use of uncleaned implements employed by consumptive persons in taking their food. It is very rare, however, to get a clear history pointing in that direction. I have only been able to get one.

I have already mentioned the danger of infection from the dust of a room occupied by a consumptive person, especially in the absence of rigorous care, and with a deficiency of light and air. It is, however, necessary to call special attention to the great risk run by the occupancy of rooms previously inhabited by a consumptive person. I have recorded a striking illustration of this danger, in which three members of a healthy family perished, apparently in consequence of the occupation of such a room in Southport.

Other instances of a like nature have also been given. This risk is very great at all our health resorts—which I think do not deserve the name, if the most rigorous disinfection of rooms is neglected after occupancy by a consumptive person, before new invalids are admitted. Owing to the state of health of many of the new occupants, the danger is very great indeed.

I here give the death-rates from phthisis of three of our most famous health resorts during the years 1881-90, not in order to show that the very high death-rates from consumption here recorded are the consequence of infection there :—

1881-90.—*Mortality from Phthisis per* 1,000 *persons.*

	25—	35—	45—	55—65
Hastings	3.59	4.47	3.66	2.19
Isle of Wight	4.70	3.98	2.64	1.83
Bournemouth (with Christchurch)	6.59	5.88	3.42	2.20
London	3.15	4.21	3.93	3.37
England and Wales	2.90	3.13	2.74	2.17

If, however, we throw over this explanation of these high death-rates, we are obliged to conclude that the excessive mortality is due to the high death-rate of consumptive visitors, and that the risk to fresh visitors must be exceedingly great unless the most stringent precautions are enforced.

I have said enough, I think, to show that the risk of infection from a consumptive person is not confined to his own family circle, but that it extends to all ranks of life; and that the rich cannot—in regard to this disease, less indeed than for any other—afford not to know how the poor live, or what their consumptive neighbour is doing.

We may now briefly consider the evidence which we possess as to the infection of man by tuberculosis in animals.

It has been said that Villemin demonstrated the identity of tuberculosis in man and in cattle. His experiments are borne out by the identical effects of tuberculin when injected respectively into human beings and oxen. Instances have also been recorded in which tuberculosis has seemed to be communicated from cattle to man by direct inoculation. Thus, Dr. Heron quotes a case given by L. Pfeiffer in the *Zeitschrift für Hygiene.* Arloing, in his book, *Leçons sur la Tuberculose*, page 349, gives two cases of accidental inoculation of veterinary surgeons, recorded by Tscherning and Nocard respectively.

The milk from tuberculous cows has been abundantly shown to be infectious, and in the case of cows with tuberculous udders, virulently infectious. After the recent report of the Royal Commission on tuberculosis, it seems scarcely necessary to labour this matter. The Report declares that while milk from tuberculous udders alone is highly infective, the onset of tuberculosis in the udder may occur at any time. The Report evidently points to the desirability of an effort to get rid altogether of tuberculous milch cows.

Pigs are frequently tuberculous, and there is sufficient evidence that this is often due to the ingestion of milk from tuberculous cows.

Horses are much more frequently found to be tuberculous than was formerly supposed to be the case. Nocard states that the primary seat of the disease is generally abdominal, though he cannot say why this is so; but I am informed by Professor Delépine that he has ascertained from veterinary surgeons that foals are not rarely fed on cows' milk: a fact which would sufficiently explain the occurrence. From an investigation of some 400 animals slaughtered recently at the Manchester Abattoir, in which the ages and primary seat of the lesion were recorded by Mr. King, the Chief Veterinary Inspector of the Manchester Corporation, it appears that in far the majority of young animals the primary seat of tuberculosis is abdominal. In the older animals, in considerably the larger number, it is pulmonary.

This can, I think, be only explained by infection of the young animal being, in a preponderating degree, through the ingestion of milk.

Thus, we have abundant reason to believe that young animals are largely infected by milk, even when the milk is not known to be infective; and I do not know that there need be any hesitation in applying these results to the

infection of children. We may assume, I think, that a widespread process of infection is going on.

There have been, no doubt, instances in which children have seemed to contract tuberculosis of the abdomen by the ingestion of milk from a tuberculous cow, and they have some value; but, as I have said, there is a massive volume of evidence outside such direct proof.

It is, however, a matter of some moment to arrive at definiteness of conception with regard to the actual amount of infectious milk in circulation. Investigations have, I am informed, been made on this point in Paris, and are being made in Liverpool, but I will content myself with quoting from the *Centralblatt für Bacteriologie*, Jan. 20, 1897:—

Out of nine samples of milk purchased in the open market in Halle, and examined by Buege, two gave positive results by inoculation in guinea pigs, but did not reveal bacilli on microscopic examination. In the same number are given the results obtained by J. Christian Bay, and published in the Annual Report of the Iowa State Dairy Commissioner, 1896, of a microscopic examination of 359 unmixed and 204 mixed samples. Of the 204 mixed samples four, or 2 per cent., revealed the presence of tubercle bacilli; of the 359, fifty-one, or 14 per cent., showed tubercle bacilli.

These are sufficiently formidable results, even if we take only 2 per cent. as a standard, since we know that now one, now another milk supply will, in this respect, undergo variations.

I have not investigated the subject from this point of view, but have sought to determine how the Manchester cowsheds stand as regards conditions which may be reasonably regarded as dangerous.

I have recently examined nine farms in Manchester with an aggregate of 108 cows. Of these, seven have presented a distinct nodular condition of the udder, presumably tuberculous. Three have been tested by Mr. King with tuberculin, of which two gave a marked local reaction, and in the third there could be no doubt about the tuberculous nature of the lesion. These two cows were slaughtered at the abattoir, and the udders taken to Professor Delépine, who found both to present well-marked tubercular changes.

In two other cases the udders have been positively diagnosed as tuberculous. Altogether, twenty-three milch cows have been slaughtered during the last year, when condemned by the veterinary inspector.

As regards milk I have these facts. Nineteen samples have been obtained and examined by Professor Delépine.

These have been taken from selected cows either on account of abdominal tuberculosis or by reason of nodular disease of the udder. Only one milk gave microscopical evidence of tubercle bacilli. Four milks have proved to be tuberculous by inoculation in guinea-pigs. The investigation is, however, incomplete. Two cowkeepers have had their herds examined by tuberculin.

We may assume, I think, that the condition of things is but little better outside the city, and that, in fact, much tubercular infection by milk is actually going on. Should anyone object that the guinea pig is an animal easy to infect by inoculation, the answer is that, unfortunately, there are vast numbers of young children, who are also in a condition, at one time or another, which renders them easy to infect.

As regards butchers' meat, we may content ourselves with referring to the recent report of the Royal Commission, which shows that there is a certain degree of danger from the consumption of tuberculous meat: a danger not removed by cooking. The Report also makes it clear that, in the ordinary processes of killing, this danger is not confined to the meat from tuberculous animals, but may be transferred to sound meat by imperfectly-cleaned instruments—a danger which exists in the butcher's shop as well as at the abattoir.

Nevertheless, having regard to the rarity of primary abdominal tuberculosis in the adult, this danger is not comparable with that attaching to milk.

The experiments made for the Royal Commission, and other facts, also show that a real danger attaches to the occurrence of the disease in domestic pets, especially in cats, and more accurate knowledge in regard to the extent of this danger is much to be desired.

I have spoken of the danger attaching to the milk of tuberculous cows, but there is another, which must not be overlooked. There is no doubt that tuberculosis introduced into a crowded cowshed, spreads rapidly from cow to cow. The greater part of the bovine discharges from a tuberculous lung is swallowed, but a certain amount escapes from the nostrils. Doubtless a great proportion of the bacilli are destroyed in the stomach of the cow, but—especially when there is much spore formation—part will be discharged in the excreta.

With the excreta, in many cowsheds, the haunches and

even the udders of the cows are plastered, and there the infectious matter dries. Of this a certain amount will certainly get discharged, and float in the air of the cowshed. It is thus no wonder that tuberculosis spreads rapidly in many cowsheds.

But the same consideration attaches to the attendants on the cows, though in a far less degree. If we look to the high phthisical death-rate in many rural districts, we cannot fail to perceive that this subject requires a profound and searching investigation. It is even a superstition, in some parts of this country, that it is beneficial to a consumptive person to be much amongst cows in the cowshed. It is, at all events, not very beneficial for the cows.

May we hope that Dr. Thorne Thorne will direct the attention of his staff to this and kindred problems.

I have not been able to obtain personal evidence of the communication of tuberculosis in this way, but two striking experiences pointing in that direction are given by Nocard in his book on the *Animal Tuberculosis* (pp. 80 and 81);* and Dr. Heron, in his work on the *Communicability of Consumption,* quotes Hedley in the same sense. These observations, however, are inconclusive, and need extending.

Adjuvant Influences in the Production of Tuberculosis.

Let us now very briefly consider the effects of various conditions which have been adduced as important influences in the causation of tuberculosis.

The usual way of approaching this subject has been to exhibit the influence of various causes in producing tuberculosis, and then to bring in the infective element as an accessory. I propose to reverse the process, and for this reason: It is very rare that we have exhausted the influences adjuvant to infection when we have enumerated one, such as occupation, and we are thus liable to an improper estimate of the factor selected. This is manifest, if we consider the high phthisis death-rates prevailing in different parts of the country under the most diverse conditions, amongst people pursuing the most different occupations.

If, however, we start with infection, we stand on firm ground.

The bacillus of tuberculosis grows slowly in the animal

* "The Animal Tuberculosis and their relation to Human Tuberculosis", by Ed. Nocard, translated by Dr. Scurfield. Baillière, Tindall and Cox, 1895.

body, and each step of growth is strenuously contested by a small army of white blood corpuscles. Sometimes the invader wins, sometimes the defender. Frequently a foothold is gained, to be lost by increased strenuousness of defence. If the air passages are intact, it is with the greatest difficulty that a limited number of bacilli or spores can pass the ciliated portion of the tract, and invasion is only successful if the invaders are very numerous.

An odd bacillus will have practically no chance, unless it happens to light upon, or be carried by a leucocyte to, an injured place.

Hence a healthy individual is not likely to succumb to tuberculosis, unless assailed by a considerable amount of infective particles.

From clinical inquiry in Oldham in 1890, one was able to formulate the proposition that *healthy persons* do not contract phthisis except after prolonged and intimate exposure to infection.

It is manifest, however, that if, as with Koch's inhalation experiments, a large number of bacilli reach the alveoli, the defending leucocytes are divided, weakened, and overpowered.

If, on the other hand, a limited number reach some abraded and injured portion of the bronchial tract, the leucocytes lying on the surface are easily overpowered, and a nucleus of growth is formed, Such is also the case with portions of the lung in which degenerative changes have occurred, or in which there is but a feeble circulation owing to imperfect expansion.

Thus the first and most important condition is abundant infection. This, however, is a condition which we know to be fulfilled in common lodging-houses, and in lodging-houses of the poorer sort, and which was formerly present in a high degree in our barracks and prisons. It is also, not infrequently, fulfilled in the homes of the poor, where the air and light necessary to dilute and destroy the infection are frequently absent.

The effect of overcrowding in weakening and predisposing the system to the reception of infection, through the inhalation of vitiated air, is not to be denied. On the other hand, Bang found a large number of herds of cows on small farms in Zeeland free from tuberculosis, many of which we may be certain were stabled in small and insanitary cowsheds. I have, within the last two or three days, come upon an experience bearing on this point. While visiting a seminary I was much struck with the

crowded appearance of the dormitories. They were, however, spotlessly clean, and I was assured that the bedding and floors were very frequently washed. They were well-lighted, and all the windows were open. As soon as a child with tuberculosis is discovered to have been admitted he is removed. Under these cirumstances, I am assured by the medical adviser to the Institution, that cases of tuberculosis do not arise among the children. The effect of crowding on the general health cannot fail, however, to be injurious.

Tuberculous cows had, apparently, not been introduced amongst the small herds mentioned above. Doubtless, once introduced, the disease would spread rapidly. But, manifestly, overcrowding will not produce tuberculosis, though it is probably the most powerful adjuvant in its spread, when once it has been introduced.

Injuries of the respiratory tract may be produced in various ways. Thus, certain occupations are specially liable to cause a condition favourable to infection: such as file-cutting, needle-polishing, flint-working, grindstone-making, stone-cutting, glass-cutting.

For the same reason, phthisis is liable to follow such diseases as measles, typhoid fever, pleurisy, bronchitis and pneumonia. Possibly the predisposing influence of alcoholism may come under this head.

In the presence of a certain degree of infection, any causes of physiological depression, whether physical or mental, is liable to be attended with the development of phthisis: the more so according to the measure of impairment of the lung which they produce.

In the production of tuberculosis, however, each adjuvant may in its turn be dispensed with, providing the fundamental requirement of infection is present; infection which must be abundant, and probably must be prolonged for the healthy, but which may in comparatively small amount affect those who have sustained injury, or who are constitutionally feeble. Unquestionably the disease spreads rapidly in crowded lodgings and homes, and the effects of overcrowding are unmistakable, to whatever factor we ascribe the result.

Let us, for the sake of example, assume that we have twenty men living at a common lodging-house, and using a small sitting-room, 12 ft. by 12 ft. by 10 ft., at the back of the house, which is overshadowed by neighbouring buildings; and let us suppose that three of them are phthisical. Amongst the men some are tramps, some labourers; there

is one moulder and one stonemason. The room possesses no special means of ventilation.

We may assume that the three phthisical men spit on the floor.

Let us first ask ourselves, is there a probability of much infection being in the air of the room? We may safely say that, with three phthisical men, there is.

Is the danger less to two men, if only two occupy the room besides the consumptives, than it would be if the remaining seventeen were present? Assuredly it is: but mainly for the reason that with so many men present there will be much more movement of dust in the room than if only two are present. That is one unquestionable reason of the influence of overcrowding in the production of tuberculosis.

Another reason is that the parts on which the infectious sputum has lighted are much more likely to be trodden on; and that it is thus almost certain to be much sooner dried and dispersed as dust with seventeen than with two men in the room.

It is, in fact, largely the movement which causes the evil effects of overcrowding as regards phthisis, and hence the day-rooms of our worse common lodging-houses are simply breeding-grounds of phthisis.

The same, however, applies to any group of people shut up in a confined space, insufficiently ventilated.

The stonemason, in such a position, would be likely to suffer soon; but it by no means follows that he would be the first, as some of the others would be liable to suffer from time to time from attacks of alcoholic catarrh.

In fact, overcrowding, foul air, want of light, want of cleanliness, injury from diseases, excess, misery, privation, etc.; injurious occupations, feebleness of constitution, and other adjuvant causes, may each assist in the implanting of infection; but not one of them is indispensable, providing that from any cause there has been a sufficient accumulation of infective matter in a dry condition. We see occasionally the most robust people succumb to intense exposure to infection; and there is but too much reason to suppose that the disease is not rarely contracted from an accumulation of infection which has taken place, beyond the ken of the sufferer, in clothing, in milk, and so forth.

Nevertheless, much may be done by rigorous cleansing with water, and by a scientific use of light and ventilation, to remove, dilute, and destroy the infecting germs.

We must not, however, in our endeavour to extend the

range of our action, forget for a moment the splendid results which have been achieved by general and indirect measures, both as regards phthisis and in the improvement of our conditions of health generally. Our beneficent factory legislation, the many betterments which have taken place in the housing of the people, and the removal of many injurious influences connected with trade, have resulted from the labours of an army of distinguished inquirers, which has been led by such men as Sir John Simon, Sir George Buchanan, Greenhow, and Dr. Farr. But it is noteworthy that the younger generation of distinguished men, who have devoted much thought to this subject, have turned largely towards a more direct handling of tuberculosis.

Two objections have been raised to a vigorous prophylaxis in tuberculosis. One of these is heredity. Another is the potentiality of growth on soils outside the body at the ordinary temperature. Supposing, even, all that could be alleged in these regards were true, it should not deter the advocate of prevention, though it might somewhat discourage him.

As regards heredity, the argument would run thus:—

Tuberculosis is an inherited disease. Therefore, it is useless to take measures to protect a large section of the community, as they have inherited the disease, which will develop whatever you do.

It will be at once seen that this argument supposes the inheritance of the tubercle bacillus. No one disputes that people with feeble circulation and weak chests are more liable to phthisis than robust people with good constitutions, cæteris paribus; and that their children, who are very liable to inherit the same weakness, are for that reason more prone than stronger persons to contract tuberculosis. In the word "stronger" we include all those peculiarities of nervous system, personal habit, etc., which make for strength, as well as mere anatomical build.

It is sufficient to state ordinary plain facts to see how unlikely it is that there is anything more than this in hereditary predisposition. If animals, which in a state of nature are absolutely free from tuberculosis, are inoculated or fed with tuberculous matter, they take the disease and succumb, the progress of the disease depending on the size and strength of the animal, and on nothing else. Is not this, of itself, almost sufficient?

If we have regard to the history of phthisis in the Foot-Guards, a selected body of men of the finest physique in the

country, amongst whom there would be certain not to be a history of tuberculous inheritance, or to the history of the selected women who suffered so terribly in the Catholic nursing institutions, or, again, to the rapid spread of tuberculosis when introduced into populations virgin to the disease, we are obliged to admit that in the absence of a hereditary tendency there is no immunity from infection.

At all events we may safely say as much as that, and when we have said that much, we have left little for a hereditary tendency to account for. Certainly, hereditary predisposition of tissue is disposed of.*

It is, however, the contention that the bacillus tuberculosis is inherited that must be met by the advocate of preventive measures, if he wishes to avoid successful opposition. The above facts are equally condemnatory of this view; which, moreover, has, as far as I can see, nothing to support it, beyond what there is to support the inheritance, for example, of small-pox.

It is impossible to maintain the view that children inherit the disease from their parents, since in the majority of instances the parents have not had phthisis when the children die of it.

If, however, we take in grandfathers and grandmothers, as Baumgarten would have us do, we have, excluding the grandchild, six persons; and it would indeed be strange if, in a large proportion of cases, one or other of the parents and grandparents of a consumptive has not had consumption, when it has been estimated that one-seventh of the human race perishes from this disease. Specially remarkable would this be when one remembers the weak physique which the parents and grandparents have often had, and the intimate exposure to infection which a child so often undergoes from a consumptive parent.

It is, however, much safer to rely on pathological facts rather than on statistics. As Koch has pointed out, authentic instances of congenital tuberculosis are by comparison very infrequent; but the pathological appearances in these instances do not at all suggest that the bacillus can be born with the child in a latent condition, and without any pathological appearance to show for its presence. It is, indeed, probable from the manner of propagation of tuberculosis inside the body, that congenital tuberculosis can only occur when a discharge of bacilli has occurred into the blood-stream: an occurrence which is itself not

* See also Sturges' facts in the *Lancet*, Nov. 17th, 1888.

frequent. But it is not necessary to rely upon this argument.

The following facts, attested by Koch and Bang, are of overwhelming weight as regards this matter. According to Koch, the offspring of tuberculous guinea pigs are nearly always free from tuberculosis, and if removed from the infected stall do not become tuberculous.

According to Bang, the calves born of tuberculous cows—cows, it is true, not in an advanced stage of tuberculosis—do not when born give a reaction to tuberculin; and when isolated from cows known to be tuberculous, do not, with very rare exceptions, develop tuberculosis.

These facts set us free to put aside the doctrine of latency, and to utilise the mass of statistics which prove that it is exceedingly rare for a calf to reveal tubercular lesions when slaughtered, although the disease rapidly increases with advancing age.

In the same manner careful pathological records show that children do not have tuberculosis at birth, with rare exceptions, and that the disease steadily increases after birth.

The possibility that the tubercle bacillus may grow outside the body is one which we have to consider carefully in connection with the etiology of tuberculosis. It must now be regarded as proved, in spite of the pronouncement of Koch, that such growth is possible at ordinary temperatures, in glycerinated media at all events.

The researches of Roux and Nocard, of Pawlowsky, of Sir Hugh Beevor, and recently of Delépine, have established this so clearly that we must accept it.

Whether we are justified in drawing the conclusion that such growth does occur in damp houses, is another matter which requires special investigation.

The statistics of phthisis do not help us much.

On the one hand we have the observations of Sir George Buchanan and of Bowditch, showing that the removal of damp has been attended with a great reduction of phthisis. On the other, we have the fact that damp districts do not appear specially liable to phthisis.*

Pathologists are similarly at variance. On the one hand, we have Cornet maintaining that damp may be directly serviceable by preventing sputum from getting dried; on the other, we have Delépine contending that a study of the natural history of the bacillus leads us to suppose that on

* See also Villemin, *Sur la Tuberculose,* cap. xi; Newsholme's *Vital Statistics,* 1889, page 121, also 129 and 130.

suitable damp soils, such for example as collections of sputum, growth may continue.

Then, we may recall that Ransome has steadily maintained that damp is favourable to the life of the bacillus.

So far as my own observations extend, I may say that there seemed to be a preponderance of phthisis in damp houses, but I am not prepared to go further. It may be that the class of people who crowd together in small dwellings are content to live in small damp houses. It may be that there is something in the contention that damp favours the spread of phthisis, but only under certain circumstances, and especially if the damp is of a nature to allow a slow growth of the bacillus tuberculosis to take place.

Professor Delépine, as I understand his paper, would favour such a view; while contemplating the possibility that a damp subsoil may also favour the retention of moisture in organic solutions spilled in the house, themselves still more favourable to the growth of the bacillus.

Damp may also act in other ways: by the depressing influence which it exerts, and by rendering the system more susceptible to invasion by infection in consequence of catarrhal attacks.

Organic damp, such as arises from midden privies, may also act as a nidus for septic micro-organisms such as those which proved so fatal in Cornet's experiments. In this way a damp subsoil may exercise a very injurious influence on health, powerfully predisposing to phthisis.

Such considerations, however, should only increase our efforts to attain cleanliness.

In fact, our contention is that we are in presence of a disease which is purely infectious, and is inherited only to a trifling extent; and that, knowing as we do the laws governing this infection sufficiently for practical purposes, It is our duty to make a determined effort to remove the infected matter. Each step makes the next step more easy.

Now, unquestionably, the first thing which we have to do is to educate all classes to take their part in an effort which must be severe, but which is by no means a hopeless one, to erase tuberculosis from the list of prevalent diseases.

EDUCATION.

The medical attendant must learn to make a well-weighed and clear counsel in regard to prevention a part of his medical treatment, as indeed the most ordinary humanity would prompt him to do. It is really, moreover, a

necessary part of medical treatment, since (as Ransome and Cornet have strenuously maintained) there is no doubt that the inhalation of extruded infected matter, after it has been dried and reduced to dust, is liable to light up new foci of disease in the injured lung, and thus to baffle his medical skill. He must himself learn never to use a spatula to any patient without at once carefully disinfecting it with water which has just been boiled. No instrument should ever be applied to the throat without prompt and vigorous disinfection following its application.

He is bound, for the sake of those still in health, to omit no means of diagnosis within his reach, in order that immediate steps may be taken for preventing the spread of the disease.

The health authority of every district, where the means of diagnosis are available, should place within reach of the practitioner the means necessary to make an early and accurate diagnosis, in so far as they are such that he may not reasonably be expected to be familiar with their use.

The health authority may profitably place at the disposal of the practioner an account of the means by which he may best aid his patients, in protecting both themselves and their intimates from further infection.

This has been done in Manchester and Salford, by means of the Memorandum of the North-Western Branch of the Society of Medical Officers of Health: a memorandum drafted by Mr. Paget.

Dr. Russell, of Glasgow, has recently placed his views at the disposal of the practitioners of Glasgow. Of course, such aid is not necessary in many instances.

The medical officers of public institutions may be expected to interest themselves deeply in the problems of tuberculosis. In particular, they may reasonably be expected to use all available means of diagnosis, and to be at great pains to ascertain, in case of death, in what part of the body the infection has first started.

The medical practitioner may be expected, in cases of tuberculosis, to give instructions relative to the use of Dettweiler's Pocket Spittoon; and to the means whereby his patient may avoid allowing discharges, whether from the lungs, bowels, glands, bones or ulcers, from getting dried. He may also be expected to give instructions how to clean out the sick room without disturbing the dust therein, and as to the necessity for frequent cleansing and airing of the sick room.

The Veterinary Surgeon has no mean part to play in the educational process.

He may be expected to investigate the manner in which animals become infected, which he has exceptional opportunities for doing.

He may be expected, also, to determine the extent to which tuberculosis prevails among domestic pets.

He will be called upon to instruct cowkeepers and stockbreeders in the means whereby they may hope to eliminate tuberculosis. He will advise as to the size, cleansing, ventilation and lighting of shippons, and as to the necessity for strict cleanliness, both of the cowshed and of the cow. Cleansing with water, as often as possible, is perhaps the greatest safeguard we possess. The discharges of the cow must not be allowed to get dry, and the cow must therefore be kept strictly clean. He will be able to indicate the uses of tuberculin, and to urge its application for purposes of isolation. He will advise as to the disinfection of cowsheds. He will advise as to the wickedness of using milk from a cow with a diseased udder. He will also advise as to the danger of employing phthisical people about a stable, and as to the risk which healthy people run in being much amongst tuberculous cows.

All employés and associations of workmen should be informed of the part which they may reasonably be expected to play in preventing industrial co-operation from becoming a source of danger to life. To every workshop should be sent special directions. Especially is this necessary where the workpeople are in confined rooms.

Employers owe it to their workmen, as well as to those who buy their goods, to take all possible precautions against infection. Notices should, therefore, be posted up, wherever the work is of a confined nature, advising that every workman afflicted with a chronic cough should carry a Dettweiler's pocket spittoon, and that he must not spit on the floor or in a pocket handkerchief. The spittoon may be emptied when he gets home, and then washed with boiling hot water.

The workpeople must not use each other's spittoons.

When there is a fire, they may spit in a piece of paper or rag, which must be forthwith burned; or such pieces of paper or rag may be dropped into an earthenware jar charged with water. These rules should not apply merely to persons known to be consumptive, but to all persons with a chronic cough.

Employers should be made to provide abundant exchange of air, and to keep all parts of their workroom cleansed with water.

Workpeople afflicted with a chronic cough should be educated not to carry a pocket-handerchief. If consumptive, they are liable to scatter a cloud of infection, every time they pull their pocket-handkerchief out.

If these principles are followed, workpeople run no risk of contracting consumption in the workshop.

As regards free exchange of air and strict cleansing, an effort should be made to carry out the above rules in all rooms used for the assembling of people.

The school hygiene of tuberculosis may be left to develop gradually. There can be no doubt, however, that many schoolrooms stand in need of proper ventilation. School boards, school managers, and teachers may be called upon to play their part in removing the conditions favourable to the spread of consumption, and enforcing the rule, "Thou shalt not spit". They may be expected also to give instruction in those proceedings which are necessary to avert danger of infection, especially as concerns free ventilation and cleanliness.

Railway companies should provide against the discharge of sputum in railway carriages.

Special hints should be drawn up for the use of seminaries, prisons, and lodging-houses.

The aid of clergymen should be invoked for the protection of public assemblies.

Special rules should be devised for the guidance of hotel-keepers, keepers of private lodging-houses, and hydropathic establishments.

The sanitary authorities of all health resorts may be expected to be specially vigilant in carrying out disinfecting measures in rooms vacated by consumptive persons, and in advising as to the prophylaxis of phthisis generally.

Lastly, the whole population should be furnished with brief and simple directions for preventing the communication of phthisis.

Instruction should be given to agriculturists, and especially to cowkeepers. Most of the farmers supplying milk to a large town can only be reached through the milk dealers. Milk dealers should therefore be furnished with a paper of instructions for the cowkeeper.

Instructions to cowkeepers should embrace the following points:—

The floor of a cowshed should be so constructed as to be

capable of being cleansed with water throughout, without leading to lodgment of moisture.

The cowshed should not be crowded. It should be freely ventilated by numerous small openings in the walls, so as to disperse the air as much as possible.

It should be well lighted.

Above all, it should be strictly clean; if possible, thoroughly cleansed with water at frequent intervals.

The cows should be kept clean. Excreta must not be allowed to dry on them.

A tuberculous cow may at any time give infectious milk, and should therefore be slaughtered as speedily as possible.

A cow with a nodular udder should be tested with tuberculin as well as by examination of the milk, and, if found to be tuberculous, should be at once slaughtered.

The farmer should be advised to have his herd tested with tuberculin. He may then gradually eliminate the unsound cows, and replace them by healthy cows guaranteed free from tuberculosis. Or he may pursue the plan adopted by Bang, whose experience warrants the expectation that tuberculosis once banished from the cowshed may be easily kept out.

The attention of the farmer should be drawn to the possibility of his milk supply being stopped, owing to the presence of disease amongst his cows.

In order to keep disease from amongst his cows, it will be necessary to exclude phthisical farm-servants.

Committees managing public institutions may be expected to invite tenders for the supply of milk guaranteed free from tuberculosis, by the tuberculin test, applied by a veterinary surgeon appointed by themselves. The Sunderland Fever Hospital is now being supplied from cows so guaranteed, after the application of the test by a veterinary surgeon appointed by the Corporation.

Medical men may reasonably be expected to urge their patients to keep up a demand for milk guaranteed free from tuberculosis.

I will not enlarge upon education further than to state that many health officers are now vigorously engaged therein, but pass on to state distinctly that, in my opinion, tuberculosis should be made a notifiable disease.

Administration. Notification of Tuberculosis.

If we are to deal successfully with the prophylaxis of this disease, we must have a full and accurate knowledge

of its ramifications; and it must not be left to here-and-there an ardent physician or surgeon to point out the dangers of communication and the means of averting them.

It must, I think, become the duty of the Public Health Service to collect and sift information, seeking aid from physicians, surgeons and pathologists, and to apply the accurate knowledge thus gained by devising practicable methods of prevention. I am fully aware of the delicacy of the task which I am proposing to lay on the Public Health Service. But surely we are to remember that in regard to no other infectious disease have we, even now, such clear conceptions as to its causation. We know where the infective matter comes from, how it reaches its victim; how, in various ways, to intercept and cut off the path of the parasite. We do but need to be brought in contact with the individuals.

It may be said that we shall thus rudely disturb many social relations, and greatly alarm many unhappy people. I have not found such to be the case in the course of my personal inquiries; not even, indeed, when inquiries have been carried out by sanitary inspectors. It has further been said that preventive measures may safely be left to the Medical Attendant. The Medical Attendant is usually a busy man, who has enough to do in dealing with the healing part of his profession; though undoubtedly he might, if he chose, do much valuable prophylactic work, and is doubtless doing so to an increasing extent. But I must say, bluntly, that this putting of preventive work entirely on the shoulders of the Medical Attendant is inadequate and unsatisfactory. Why, were it nothing else, it is most essential that the sanitary conditions, where phthisis is abundant, should be investigated and removed.

It is even most necessary that the Health Authority should be made aware of the enormous amount of mischief which results from overcrowding, and other insanitary conditions.

However, I admit that in many instances much tact would be required. I am sure that the Medical Attendant and the Medical Officer of Health in combination would be able to straighten out the difficulties which attach to any administrative scheme resulting from notification.

The diseases which would be notified would be all forms of tuberculosis attended with discharge, and also cases of tabes mesenterica.

Notification of the latter is necessary to enable us to

obtain sufficient evidence to justify us in examining herds of cows, from which we may be able to trace milk infection.

Notification, to be of any real use, must be compulsory, and a fee must be paid as for all other notifiable diseases—I have elsewhere shown that we need not be deterred on the score of expense. Otherwise, I think the understanding as to the applications of notification, on the strength of which the Oldham Medical Society were prepared to recommend notification to the sanitary authority, would make a tolerable basis on which to start.

The position taken up by the Oldham Medical Society was the following:—

Extract from the *Lancet*, 1893, Vol 2, p. 1257:—

"*Oldham Medical Society*—On October 24th a discussion on the subject of Notification of Phthisis was opened by Mr. Fawsitt, president of the Society, at a very full meeting. Several members took part in the discussion, and at the close of the meeting the subject was adjourned to another meeting, which was held on November 7th. A number of speakers having expressed their views on the question, it was understood that the objects of notification would be: (1) The sanitary authority would disinfect the room occupied by the tuberculous patient, as might be required. (2) Inasmuch as the transmission of tuberculosis is, in a large proportion of cases, determined by the presence of insanitary conditions, such as damp, overcrowding, deficiency of light, and so forth, an opportunity would be afforded to the authority of removing such conditions, and so saving other members of the household. (3) Further evidence would be collected, bearing on the modes and conditions of transmission of the disease. The personal hygiene of the patient would in no way come under the care of the sanitary authority; it was hoped, however, that the medical attendant would confer with the medical officer of health, when convenient, as to the precise means by which tuberculous discharges might be most usefully destroyed. At the same time, the sanitary department would in no way interfere with the treatment of the patient, and all instructions in dealing with discharges from the patient would come from the medical attendant. (5) Only cases would be reported in which phthisical expectoration was taking place from the lungs, cases with tuberculous diarrhœa, and cases with copious discharge from tuberculous bones and tuberculous ulcers. These and other matters having been fully discussed, the following resolution was carried unanimously:—

"'That the Society, having had its attention drawn to the infection of tuberculous diseases, and to the great mortality and injury to the population arising therefrom, is of opinion that these diseases should be the subject of voluntary notification to the sanitary authority.'

"It was understood that a copy of the resolution would be sent to the Sanitary Committee of Oldham, as well as to the medical journals."

It is needless to enter on the subject of isolation of cases of tuberculosis in hospital at present. When we have once obtained notification of the disease, the administrative measures which arise therefrom may be left to develop.

As regards the dangers arising from tuberculous milk and tuberculous meat, I do not propose to say very much, especially as regards tuberculous meat. The chief requirement in respect of tuberculous meat is that medical officers of health and veterinary surgeons should be trained to recognise tuberculosis in meat; that is to say, where to look for tuberculous glands and how to recognise them. No meat should be placed on the market without examination; and, as the law is at present administered, it may be taken that sanitary inspectors also should undergo a course of training in the more familiar conditions rendering meat dangerous as food.

Some system should be devised under which a cowkeeper is not tempted to retain a tuberculous milch cow for the production of milk; but is rather encouraged, and even urged, to slaughter tuberculous cows as soon as possible, under proper control.

There can be no doubt, from recent observations, that tuberculous udders are by no means of rare occurrence, although all nodular udders cannot be so classed.

The use of tuberculin, which is eminently adapted to clear up the nature of these nodules by the local reaction produced—a purpose for which I have had it used recently in Manchester—should, I think, be regarded as compulsory in these cases.

Tuberculin may be said to have two chief applications at present. One is where the diagnosis of advanced tuberculosis is made by clinical examination, or where a nodular udder is present. In such cases, it should be regarded as compulsory, and as a proper mode of completing the diagnosis. If a cowkeeper declines, under such circumstances, to have tuberculin used, he has only himself to blame if a mistake has been made.

The other use is a voluntary one, where the cowkeeper

desires to separate his sound from his unsound cows, and gradually to eliminate the latter. For this purpose, he should be aided by the services of a public veterinary surgeon, empowered to apply tuberculin at the public expense, and by the advice of the Medical Officer of Health as to how he may carry out isolation and disinfection.

In the absence of the tuberculin test, samples of every pailful of milk from each farm may be tested for the presence of tubercle bacilli, and the cows should be systematically examined by a veterinary surgeon in every district.

This means the establishment of a Veterinary Public Health Service.

Uniform regulations as regards cleanliness, lighting, and ventilation of cowsheds should be enforced.

The Medical Officer of Health should be empowered to order the slaughter of any cow shown to yield tuberculous milk. At present, he can only forbid such milk being added to the general milk supply. It is true that this provision of the Order of 1885 is meaningless, unless he can cause the cow to be removed from the cowshed; in which case it must either be allowed to go dry, or used to feed a calf, or slaughtered.

As regards the milk supply outside his district, he is at present comparatively powerless. Doubtless, if a sample of milk from the general supply is shown to be tuberculous, and if he can ascertain the cowkeeper from whose farm the milk has come, he may, at considerable trouble, visit the farm along with a veterinary surgeon and examine the cows. Or he may stop the milk supply for a time.

Samples should be taken of milk arriving in towns from separate farms, for examination for tuberculous infection, either at the station or at the dealer's.

Milk dealers should be required to assist in tracing infection. For that purpose they should, as far as possible, confine the supplies from individual farmers to particular households, and should keep a daily list of households supplied from the respective farms.

Samples of milk from individual cowsheds should be, for each district, submitted to an expert bacteriologist, and, when possible, from each suspicious animal.

By these means we shall be able to say definitely whether certain cows are infective, and must be removed from the cowshed. In that case a substantial guarantee of the disuse of the animal from supplying milk for human consumption should be afforded. The cow should be

slaughtered, or fatted and let go dry; or used to feed a calf for slaughter. But the last two courses are unsatisfactory.

Bang advocates using cows not highly tuberculous for the production of calves. Such calves are not born tuberculous, and do not, if properly isolated, become so.

Tubercle bacilli are not distributed equally in milk supplies; and we might find, apart from any change in the cows, that the same milk supply next day revealed no bacilli. We must, therefore, adopt the more troublesome course of action, of visiting the cowsheds and examining the cows, unless some arrangement can be made.

Similarly, in a well-authenticated case of tabes mesenterica, the Medical Officer of Health may find good reason for suspecting some definite milk farm, which he would then have good cause for visiting. This is a cumbrous proceeding.

The Medical Officer of Health and the Veterinary Surgeon will probably find that they cannot possibly do the work, and these provisions do not sufficiently meet the requirements of towns.

The only way of really grappling with the problem is to have the cows examined and tested with tuberculin from time to time by a competent veterinary surgeon, who should be accountable to a central authority, such as the County Council. It should be possible to get rid of tuberculosis, and to keep it out of the cowshed, by systematic cleansing of cows and cowsheds, and by other means.

Powers should be obtained to oblige landowners to construct farm buildings in accordance with the model regulations recommended by the Local Government Board.

All medical men should urge their patients to demand milk from cows guaranteed free from tuberculosis by the tuberculin test, and to sustain their demand until this condition is complied with.

All public institutions should insist on having such milk guaranteed by their own veterinary surgeon, if necessary. Veterinary surgeons should put forward the urgency of this requirement.

Such milk would be more expensive for a time, but would afterwards be cheaper than milk now is. This is evident, if we consider the loss in value of a milch cow which has to be fatted and slaughtered.

I have dwelt at somewhat greater length on this subject than on the question of meat, which from the point of view

of prevention of tuberculosis in man, is no doubt of very much less importance.

In conclusion, man contracts tuberculosis chiefly by infection from his fellow man. The first step necessary in dealing with this source of infection is to make tuberculosis a notifiable disease.

The next most serious source of infection is cattle; chiefly by means of milk so far as our information at present goes, but also, no doubt, partly by means of the atmosphere of contaminated stables to an uncertain extent, which needs investigation.

Here we require a veterinary service, and a continued effort to eliminate the disease altogether.

There are also, however, other sources of infection which still require elucidation, whether by medical or veterinary pathologists does not much matter. Especially do we need accurate knowledge about cats, and other household animals.

I have already explained how it should be possible, by the combined application of clinical examination of the microscope, of tuberculin, and of post-mortem examination, to attain much more accurate knowledge in regard to the disease than we at present possess—knowledge which would be invaluable for preventive purposes.

SOME OBSERVATIONS ON THE INFECTIVITY OF DIPHTHERIA, AND ON THE RELATION OF DIPHTHERIA PREVALENCE TO SCHOOL-CLOSURE.

By LOUIS PARKES, M.D., D.P.H., M.O.H., Chelsea.

(*Read before the Epidemiological Society, April 30th,* 1897.)

Mr. President and Gentlemen,—The epidemiology of diphtheria is still so obscure, that any observations bearing on the subject, however partial and inadequate, are certain to be of interest to this Society.

In London and some other large towns diphtheria has for many years past been endemic, but with occasional large localised increases of prevalence—what may be called epidemic extensions—and it is my object this evening to contrast the behaviour of the disease in Chelsea, my own district, during what we may consider its normal prevalences and its epidemic prevalences.

In introduction, I may say that these remarks apply to Chelsea home district, which during the seven years under review—1890 to 1896—had an average population of 74,800; the increase in this period being a small one of less than 1,000. Kensal Town, the outlying district of Chelsea, has been excluded, as it geographically forms another portion of London, and its epidemic visitations are more closely associated with those of Paddington and Kensington, which adjoin it, than with Chelsea.

During the first five and three-quarter years, namely, 1890-1-2-3-4, and up to the end of September, 1895, there prevailed what may be termed a normal prevalence of diphtheria—the number of cases being as follows in each year: 1890, 172 cases; 1891, 155 cases: 1892, 164 cases; 1893, 201 cases; 1894, 185 cases; 1895, to Sept. 30, 148 cases. In the last quarter, however, of 1895, the number of cases rose to 126, and continued high in January, 1896. The prevalence then diminished until late in the spring, when cases of the disease again increased in frequency.

The disease appeared in epidemic form in July, and continued so practically until the end of the year. The number of cases occurring in the five months, July to November, reached the high total of 264, that is to say, more cases occurred in these five months than in any previous entire year since notification commenced (1890), except 1895, when the total was 274.

The especial features which I wish to bring to your notice, in the epidemic and non-epidemic periods respectively, are the relative occurrences of secondary or multiple cases in houses, the age incidence of the disease, and the case mortality.

The non-epidemic period comprises the five years, 1890 to 1895 inclusive; 1895, from January 1st to September 30th, and 1896, from February 1st to June 30th. The epidemic period comprises from October 1st, 1895, to January 31st, 1896, and from July 1st, 1896, to November 30th, 1896—a period of nine months in all. The multiple cases are cases of diphtheria occurring in houses within one month of the first case notified, and accordingly include the primary cases in all instances, as well as the so-called secondary cases. With regard to these cases, it may be said that, although a month has been taken as the period elapsing between the first case notified and the others that may succeed it, in a very large majority the interval of time elapsing between the notifications is a very short one—only a few days—and in a considerable proportion the notifications were practically simultaneous. I have not employed the term "secondary" in these cases, as it appears to involve the suggestion that they are consequent or dependent upon the primary case; whereas it is quite possible, owing to delays in diagnosis and notification, and the uncertainty of the exact period of incubation in diphtheria, for some of these multiple cases to have contracted the infection simultaneously from the original source, which was the common source of origin of these cases.

In the non-epidemic period of six years and two months, 1,153 cases of diphtheria were notified; and it is as well to add here, that diphtheria throughout this paper includes all cases of membranous croup. Of this number 209, or 18.1 per cent., were multiple cases as defined above. During the epidemic period of nine months, 421 cases of diphtheria were notified; and of this number 168, or 39.9 per cent., were multiple cases. It is evident, therefore, that there were in proportion more than twice as many multiple cases in the epidemic period than in the non-epidemic period. In

neither period have there been included in the multiples, cases occurring in hospitals, workhouses, infirmaries, and residential schools, but only those cases occurring in ordinary dwelling-houses.

The following tables give particulars as to the numbers attacked in the invaded houses in the two periods :—

Of the 209 multiple cases during the non-epidemic period—

10 or 4.8 per cent.	of the total	were cases where	5	in a house	were attacked.
16 or 7.7 per cent.	,,	,,	4	,,	,,
36 or 17.2 per cent.	,,	,,	3	,,	,,
147 or 70.3 per cent.	,,	,,	2	,,	,,

Of the 168 multiple cases during the epidemic period—

15 or 8.9 per cent.	of the total	were cases where	5	in a house	were attacked.
28 or 16.7 per cent.	,,	,,	4	,,	,,
39 or 23.2 per cent.	,,	,,	3	,,	,,
86 or 51.2 per cent.	,,	,,	2	,,	,,

These figures clearly show that during the epidemic period the liability of members of the same family, or of persons residing in the same house, to contract the disease, was considerably greater than in the non-epidemic period; and can, I think, only be explained on the assumption that during the epidemic period the infectivity, and what may be termed, the "striking" power of diphtheria, was notably higher than in the non-epidemic period. There may, of course, during epidemic periods, be an altered susceptibility of the population to attack, or an altered receptivity to the lodgment of the virus; but of this we can know little or nothing.

The following is a concrete instance of the exceedingly infectious character of the disease during the recent epidemic. Between the 21st and 24th October, five persons resident in the family of a gentleman, occupying a detached house in a good neighbourhood, were notified as having diphtheria, and removed to the London Fever Hospital without delay. One case, a child of two years, proved fatal. Of the rest, one of the cases returned home on November 24th, and another on December 3rd. In both these cases repeated bacteriological examinations were made, and found to give negative results, before discharge from hospital. On December 19th, two of the sons of the gentleman returned from the country from school. Both were attacked with diphtheria within a few days of returning home. The whole house had been thoroughly disinfected at the end of October, and every precaution taken to prevent any return of the disease; but I under-

stand that after the return from school of these boys, some toys were brought out, which the children first attacked had played with, and which had been put away without disinfection into the box.

In conclusion, the figures above set out seem to indicate that during non-epidemic periods in London, there is but little tendency for diphtheria to spread in invaded houses. In epidemic periods, however, the tendency to spread in invaded houses, or for residents in a house to fall victims to a common source of infection, is very much enhanced. The removal of patients to hospital does not appear to affect the results as thus stated in any very considerable degree. The primary case is, and can be, but very rarely recognised, notified, and removed to hospital at a sufficiently early stage, to prevent all passage of infection to others in the house.

Now, as regards the fatality of the disease in the epidemic and non-epidemic periods, there were in the epidemic period 96 deaths amongst the 421 cases, giving a case mortality of 22.8 per cent. In the non-epidemic period the case mortality was only 19.3 per cent. (1,153 cases with 222 deaths). The case mortality of the single and multiple cases in each period is shown as under :—

Non-Epidemic Period.

944 single cases with 176 deaths ; Case Mortality = 18.6 per cent.
209 multiple „ 46 „ ; „ = 22.0 „

Epidemic Period.

253 single cases with 54 deaths ; Case Mortality = 21.3 per cent.
168 multiple „ 42 „ ; „ = 25.0 „

These figures appear to indicate that not only is diphtheria, unlike scarlet fever, a rather more fatal disease when epidemic, than when comparatively quiescent, but also that in both periods, multiple cases are attended with more deaths than single cases, and that the mortality of multiple cases is especially striking in epidemic periods. These case mortalities, however, have to be considered in connection with age incidence, a fatal result having, as we know, a very direct relation to the age of the patient.

The following Table shows the age incidence in the epidemic and non-epidemic periods respectively; and the case mortality, from which it appears that in the epidemic period 74.3 per cent. of the cases were under ten years of age, as against only 56.7 per cent. of the cases in the non-epidemic period. The case mortality of cases under three years of age is 9 per cent. less in the epidemic period than

in the non-epidemic, but this is balanced by a higher case mortality in the former period of cases between three and twenty years. On the whole, and having regard to the fact that the larger number of cases in the non-epidemic period occurred prior to the introduction of the anti-toxin treatment, whilst many of the cases in the recent epidemic period undoubtedly benefited by this treatment, it would appear that the tendency to a fatal issue in diphtheria is increased when the disease is epidemic, especially at the school ages.

Diphtheria in Chelsea: Age Incidence.

	Under 3 years.	3-5 years.	5-10 years.	10-20 years.	Over 20 years.
Non-Epidemic Period.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Cases (1,153) ...	171=14.8	191=16.6	291=25.3	223=19.3	227=24.0
Deaths and Case Mortality ...	87=50.9	63=33.0	48=16.5	14= 6.3	10= 3.6
Epidemic Period.					
Cases (421) ...	74=17.6	86=20.4	153=36.3	55=13.1	53=12.6
Deaths and Case Mortality ...	31=41.9	32=37.2	29=19.0	6=10.9	1= 2.0

The following Table gives an analysis of cases, deaths, and case mortality at five age groups in the two periods under review :—

Diphtheria in Chelsea: Analysis of Cases and Deaths at 5 Age Periods.

		Under 3 years.	3-5 years.	5-10 years.	10-20 years.	Over 20 years.
Non-Epidemic Period.		Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Single Cases	Cases (944) ...	136=14.4	146=15.5	230=24.3	183=19.4	249=26.4
	Deaths and Case-Mortality	68=50.0	47=32 2	40=17.4	12= 6.6	9= 3.6
Multiple Cases	Cases (209) ...	35=16.8	45=21.5	61=29.2	40=19.1	28=13.4
	Deaths and Case-Mortality	19=54.3	16=35.6	8=13. 1	2= 5.0	1= 3.6
Epidemic Period.						
Single Cases	Cases (253) ..	34=13.4	51=20.2	96=37.9	33=13.0	39=15.4
	Deaths and Case-Mortality	12=35.3	18=35.3	19=20.0	4=12.1	1= 2.6
Multiple Cases	Cases (168) ...	40=23.8	35=20.8	57=33.9	22=13.1	14= 8.3
	Deaths and Case-Mortality	19=47.5	14=40.0	10=17.5	2= 9.1	0
Whole Period, 1890-6.						
	Cases (1,574)	245=15.6	277=17.6	444=28.2	278=17.6	330=21.0
	Deaths and Case-Mortality	118=48.2	95=34.3	77=17.3	20= 7.2	11= 3.3

This Table shows that the single house cases in the epidemic period show a greater incidence rate on the ages three to ten, and a lesser incidence rate on the ages above ten years, than the similar cases in the non-epidemic period. The multiple cases in the non-epidemic period also show a higher incidence rate on all ages under ten than the single cases in the same period; whilst in the epidemic period the increased incidence of the multiple cases, as compared with the single, falls almost entirely upon ages under three years.

The case mortality of the multiple cases in both periods is seen to be higher under five years, and lower over five years of age, than the case mortality of the single cases in the similar periods. And contrasting the multiple cases in the two periods, the case mortality under three years is higher in the non-epidemic period; whilst between three and twenty years, the case mortality in the epidemic period is notably higher than in the non-epidemic.

It is interesting to note that the diphtheria that has occurred in the present year, 1897, since the decline of the epidemic, has been attended by a considerably lower case-mortality than marked the epidemic period of 1896. The disease appears to have assumed a milder type. During the seven months, June to December 1896, there were 321 cases with 72 deaths, giving a case-mortality of 22.1 per cent. During the four months, January to April 1897, there were 83 cases with 13 deaths, giving a case-mortality of 15.7 per cent.

The following Table is of interest as showing the percentage of cases of diphtheria under three years of age, at school age, or three to thirteen years, and over thirteen years, in each of the seven years 1890-6 :—

Year.	Total Cases.	Under 3 Years.	3-13 years.	Over 13 years.
1890	172	27 = 16 per cent.	71 = 41 per cent.	74 = 43 per cent.
1891	155	17 = 11 ,,	64 = 41 ,,	74 = 48 ,,
1892	164	26 = 16 ,,	69 = 42 ,,	69 = 42 ,,
1893	201	22 = 12 ,,	101 = 50 ,,	78 = 38 ,,
1894	185	22 = 11 ,,	121 = 66 ,,	42 = 23 ,,
1895	272	40 = 15 ,,	166 = 61 ,,	66 = 24 ,,
1896	428	87 = 20 ,,	269 = 63 ,,	72 = 17 ,,

It will be seen from this Table that during the first three years—1890-2—the percentage of cases at ages over thirteen was high, especially in 1891, when there was a tendency amongst certain medical men practising in the district to

notify as diphtheria in adults cases which would hardly be so diagnosed at the present time. In 1893 the percentage of cases at school age rose to 50, in 1894 to 66, with a great reduction in cases over thirteen years, and in 1895 the cases under thirteen years of age formed 76 per cent. of the total. In 1896, when there was epidemic prevalence, the cases under thirteen years formed 83 per cent. of the total, 20 per cent. being cases under three years, and 63 per cent. cases at school age.

The relation between school closure and diphtheria prevalence in Chelsea is shown in the following Table, in which are set out the average number of cases occurring at all ages, and at the school ages of 3-13 years, in 13 4-weekly periods, during the five non-epidemic years, 1890 to 1894 inclusive. The percentage which the school-age cases form of the total cases is also shown for each period.

Diphtheria in Chelsea: Average Number of Cases in 4-Weekly Periods. 1890-4.

	Total Cases.	Cases at ages 3-13.	Percentage of Total.
Weeks 1-4	8.0	3.8	47.5 per cent.
„ 5-8	11.4	4.8	42.1 „
„ 9-12	10.8	5.2	48.1 „
„ 13-16	11.4	5.6	49.1 „
„ 17-20	13.6	5.6	41.2 „
„ 21-24	11.8	5.4	45.7 „
„ 25-28	16.4	7.4	45.1 „
„ 29-32	14.8	8.2	55.3 „
„ 33-36	9.6	3.0	31.2 „
„ 37-40	14.2	7.0	49.3 „
„ 41-44	13.8	7.8	56.5 „
„ 45-48	21.2	11.8	55.7 „
„ 49-52	15.0	8.8	58.7 „

The closure of schools for the summer vacation (lasting a month), which usually commences in the 30th week of the year, is clearly followed by a fall of notified cases in the period embracing the 33rd to the 36th week. The proportional diminution is much greater amongst the children of school age than for all ages; and it will be noticed how markedly the percentage of school age cases drops in this period. There is a similar fall in cases at the end of the year, corresponding to the Christmas vacation (lasting a fortnight), but at this season of the year the prevalence of the disease is naturally decreasing—not increasing, as probably would be the case in July and August if there were no school holidays. The diminution of cases is greater in the period comprising the 33rd to the 36th week, than in the period 31st to 34th week

or 32nd to 35th week. The reason of this delay in the fall of notifications is probably to be accounted for by the period of incubation of the disease (several days), and some delay in notification, owing to difficulties in arriving at an exact diagnosis. The schools reopen usually in the 35th week of the year, so that at least for a week after school term recommences, the reduced number of notifications is noticeable, as indeed might be expected. The period embraced between the 37th and 40th week is, however, a period of markedly increased diphtheria.

In the year 1895, the diminution of diphtheria commenced simultaneously with the breaking up of the school; and the smallest number of cases were recorded in the 31-34 four-weekly period.

Diphtheria in 4-Weekly Periods, 1895.

Weeks.	Total Cases.	Cases at ages 3-13.	Percentage of total.
19-22	16	9	56.2 per cent.
23-26	16	12	75.0 „
27-30	33	17	51.5 „
31-34	11	6	54.2 „
35-38	20	14	70.0 „
39-42	27	21	77.7 „

In this year, 1895, a considerable number of cases, of which only about a half were of school age, occurred in the middle of July. A decline had already set in before the end of the month, and the effect of the school closure was apparently to accentuate this decline. In September, after the reopening of the schools, there was no immediate aggravation of the disease, only eighteen cases being notified in this month, a similar number to that notified in August. In October the disease assumed a more epidemic form, 38 cases being notified in the month, whilst in November the disease underwent a further extension, 47 cases being notified.

Weekly Notifications of Diphtheria in 1896.

Weeks.	Total Cases.	Cases at ages 3-13.	Percentage of Total.
29	7	5	71.4 per cent.
30	16	9	56.2 „
31	16	13	81.2 „
32	16	11	68.7 „
33	15	9	60.0 „
34	6	4	66.6 „
35	11	7	63.6 „
36	19	11	57.9 „
37	19	13	68.4 „
38	19	14	73.7 „
39	17	11	64.7 „

In 1896, when diphtheria was epidemic, and the epidemic was increasing in intensity, the effect of school holidays on the total cases was comparatively slight, and was confined entirely to one fortnight in August, namely, the 34th and 35th weeks of the year.

The experience of the past year appears, then, to indicate that when diphtheria is epidemic in a neighbourhood, has already got a firm foothold, and the epidemic is rising, the infection of the disease is already so widely diffused, that school closure for a limited period, such as a month, is comparatively inoperative to arrest its progress. Under such circumstances we have not only to deal with a widespread diffusion in the houses of the district affected, but also in all probability with a virus in an abnormally high state of activity, and with enhanced infective powers.

The increase in the proportion of cases at school age to total cases, as the disease became more and more prevalent in 1896, is well set out in the following figures: In February, March, and April, 1896, when the notifications were by no means excessive, the school age cases formed only 52 per cent of the total cases (27 out of 52). In May, when the increase commenced, they formed 58 per cent. of the total (14 out of 24); in June, 69 per cent. (18 out of 26); in July, 76 per cent. (42 out of 55); and in August, when the schools were closed, 63 per cent. (37 out of 59); rising again in September, after the reopening of the schools, to 66 per cent. (46 out of 70).

There is another circumstance pointing to the possible influence of the schools in aiding the dissemination of the disease in June and July last year. During the latter half of June and the whole of July out of 50 children of school age attacked with diphtheria, no fewer than 20 (= 40 per cent.) were girls or infants attending one particular Board school, which may be designated the M. R. school. Eleven of these children were in the girls' department of the school and nine were in the infants' department (three boys and six girls).

Nos. 11, 12 and 13 in the following Table lived in the same house, and Nos. 18 and 19 were brother and sister living in one house. No. 20 was a sister of No. 14, and it will be noted with regard to No. 20, that the date of her last attendance at school is 13th July, although she was not notified until the 25th July. Her sister, however, continued to attend school until the 16th July, and was notified on the 20th. It was not until the very end of the month that a pupil in the boys' department of the school

was affected, although the girls' and infants' departments were attacked in nearly equal numbers.

Diphtheria Cases attending the M. R. Board School from the Middle of June to the End of July.

Nos.	Sex.	Age.	Date of Last Attendance.			Date of Notification.
			Boys' Dept.	Girls' Dept.	Infants' Dept.	
1	F.	8	—	17 June	—	20 June
2	F.	6	—	—	23 June	24 „
3	F.	10	—	24 June	—	26 „
4	F.	3	—	—	1 July	3 July
5	F.	7	—	—	1 „	5 „
6	F.	5	—	—	2 „	5 „
7	F.	10	—	2 July	—	6 „
8	M.	7	—	—	3 July	7 „
9	F.	7	—	—	6 „	10 „
10	F.	11	—	10 July	—	16 „
11	M.	7	—	—	10 July	17 „
12	F.	12	—	14 July	—	17 „
13	F.	5	—	—	17 July	17 „
14	F.	7	—	16 July	—	20 „
15	F.	8	—	17 „	—	20 „
16	F.	8	—	17 „	—	23 „
17	F.	8	—	16 „	—	24 „
18	M.	6	—	—	16 July	24 „
19	F.	11	—	23 July	—	25 „
20	F.	9	—	13 „	—	25 „
21	M.	9	22 July.	—	—	27 „

Eliminating the multiple cases, and taking first sufferers in a house only, there were during the period under review 16 attacks amongst girls and infants attending the school. The average attendance at school of these two departments during the seven weeks in question was 635, so that the diphtheria attack-rate of first sufferers was 2.52 per cent. The attack-rate for a similar period of children of school age in the affected district, not attending the girls' and infants' department of the M. R. school, was probably not more than 0.37 per cent., or about one-seventh of the attack-rate on the girls and infants of the M. R. school. The figure 0.37 is computed from an estimate of the number of children of school age in the district chiefly affected during the latter half of June and during July, this district forming practically the central portion of the parish, and having a population of about 30,000. The total number of first sufferers amongst these children (about 5,300 in number) was 33 during the period in question; so that the first sufferers who were attending the girls' and infants' departments of the M. R. school formed 48.5 per cent. of the total number of first sufferers

in the affected district; although the girls and infants attending this school (under 700) formed only about 12 per cent. of the total number of children of school age in the district.

As bearing on the immunity of the boys of the school from attack, it is of especial interest to note that the boys have a separate school entrance in a different street from the entrance for girls and infants, and they also enjoy a separate playground. The boys at this school would appear not to mix at all with the girls and infants, either on the school premises, or in the streets adjacent. For the girls and infants, however, there is a common entrance to the school, and only one small and inadequate playground. The play hours of the two departments are kept apart as far as possible; but there must necessarily be very considerable commingling of the girls and infants when entering and leaving school, and the lavatories are also used in common.

The following Table shows the numbers of primary cases of diphtheria in newly-invaded houses in June, July, and August, in the special district around the M. R. Board School, which chiefly supplies children to the school.

North Central Area. Primary Cases in Newly-invaded Houses.

	School Age Cases.	Not School Age Cases.	Total.
June	8	5	13
July	22	6	28
August	9	7	16

It is evident from this Table that the closure of the schools in August in this special district (the north-central portion of the parish) was co-incident with a marked decline in diphtheria notifications of first sufferers at school ages in newly-invaded houses.

In other portions of the parish, however, the reverse was the case, especially in West Chelsea (west of Park Walk and Milmans Street), where the disease rapidly extended in August notwithstanding the closure of the schools.

West Chelsea. Primary Cases in Newly-invaded Houses.

	School Age Cases.	Not School Age Cases.	Total.
June	1	2	3
July	1	1	2
August	9	4	13
September	10	6	16

Having regard to all the facts, the undue incidence

on children attending the girls' and infants' departments of the M.-R. school in the six weeks ending the last week in July is very striking, and highly suggestive of this particular school being in some degree concerned in the spread of the disease at a very critical time of the year. The decline of the disease in August in the special area around the school tends to confirm this view.

In this instance, as in so many others, it is probably the continued attendance at school of a child, or children, suffering from the disease in its earlier, or in its milder and less recognisable forms, that is responsible for a certain proportion of the mischief. I do not think, however, that too much importance should be attributed to this factor. If, as I believe, the disease is generally spread by direct transference of the virus, is it obvious that during school hours only a limited diffusion is possible from an infected child to others around, even in the case of a crowded schoolroom.

In all probability, diphtheria is spread by an unrecognised case with even greater facility whilst the children are intimately commingled during their games in the hours of play in the school playground, or in the streets when assembling for or dispersing after school, than when in the classroom under the observation of the teacher. It is, I think, to a considerable extent a question of opportunities for personal contact and personal infection.

If this be so, we have some kind of indication of the reason why school closure in August is so generally followed by a diminution of diphtheria. During the holidays there is not nearly so much commingling of the children from different streets and neighbourhoods. Some of the children leave town for a fortnight's holiday in the country, the rest are content to play about in the streets, courts, or squares of their own immediate neighbourhood. The child who is in an infective condition is still, no doubt, a centre for the spread of the disease to those susceptible children with whom he may come in contact; but the size of the circle over which the infection operates is very much diminished. Hence the diminution in the number of new cases of the disease in August. The school is the effective cause for bringing the children over a considerable area into more or less intimate association with one another, and in this manner may tend to the diffusion of infection; but the facts hardly warrant the assertion that the disease is actually caught, in every instance, within the walls of the class-rooms. A careful

consideration of all the circumstances leads one rather to the belief that the orderly behaviour and restrained movements of the class-room are less effective in the transmission of infection than the violent "romping" movements and close personal contact of the children when at play.

Now, there is an influence at work in London in June and July, which undoubtedly may be an incentive to the concealment of the milder cases of the disease at this time of year. I allude to the operations of the Children's Country Holiday Fund. In July, long lists of houses, from which it is proposed to send children away to the country for a fortnight's holiday, are sent to the Medical Officers of Health of metropolitan districts, who are desired to mark off the houses in which cases of infectious disease have recently occurred. The children from these marked houses lose their country holiday. It is quite evident that a knowledge of this procedure on the part of those organising the Fund, by the parents of those children, who at this particular time are unwell or ailing, will be an inducement not to call in medical advice for fear of the prospective loss of the country holiday to children in the house. It would be interesting to know whether similar increases of diphtheria in July, of recent years, have been observed in other parts of London besides Chelsea. I have no desire to overrate the importance of the possible incentive to the concealment of mild cases by the operations of the Country Holiday Fund, which is engaged in a very useful, charitable work; but it appears to me that it is a factor that metropolitan Medical Officers of Health may have to reckon with.

Those who, as Medical Officers of Health, have to deal with diphtheria in metropolitan districts or in large provincial cities, will, I think, become more and more convinced, as time elapses, that the actual spread of diphtheria in large centres of population—apart from the question as to what are the factors tending to produce epidemicity—is almost entirely a matter of personal infection, and probably in a majority of cases a matter of direct transference of the virus from the infected to the healthy. By direct transference, I mean what takes place when children cough into each others' faces when at play, or make excessive respiratory efforts when romping or struggling together; when children kiss each other, wipe their slates with saliva, and clean their slates with their fingers; pass sweets from mouth to mouth, use a common drinking-

cup, spoon, or fork; blow their noses and wipe their mouths and expectorate into a common handkerchief, and use a common towel after their ablutions. All such acts are matters of daily occurrence in the lives of the poor; and the sooner we recognise the fact that it is personal infection that is chiefly, if not entirely, responsible for the spread of diphtheria in towns, and that the infectivity of the disease, owing to the operation of collateral circumstances with which we are but very imperfectly acquainted, is sometimes very great and at other times comparatively mild, the better position we shall be in to concert those measures which are really likely to be effectual in controlling a disease of this character.

It appears to me, that inasmuch as the elementary schools undoubtedly may at times exercise a certain influence tending to the spread of diphtheria, certain obligations might reasonably be laid upon the schools, having for their object the prevention of school attendance of those children suffering from the disease, or suspected to be so suffering. The Code of Regulations of the London School Board on the subject are very well adapted to secure these objects, but I am unaware of any similar code framed by the voluntary or church schools.

Firstly, I think it should be obligatory on the head-masters of all elementary schools, on receiving a request to that effect from the Medical Officer of Health of the district in which the school is situated, to take steps to exclude from school all children who are suffering from sore-throat. Either a medical examination of the children at the school should be made by a medical practitioner on behalf of the school authorities, or by the Medical Officer of Health himself, if he should desire it; or children attending the school, who appear to be ailing or not in their usual health, should be questioned as to the existence of sore-throat, and those who make an affirmative reply as to the existence of sore-throat should be sent home. It should also be obligatory on the head-master, when so requested, to send daily a list of the children so excluded to the Medical Officer of Health, together with their addresses. It would then be possible for the Medical Officer of Health, either himself or by means of assistants, to investigate the cases excluded, and to take such steps as might appear to him to be necessary to isolate the cases, disinfect the premises, and exclude from school other children in the house. For the better recognition of the disease in these domiciliary visitations, it would probably

be found valuable for the medical officer to be provided with the culture outfit necessary for securing some of the throat secretion in doubtful cases, which should be submitted later on to the bacteriological examination for the detection of the Klebs-Lœffler bacillus.

The utility of these methods of school exclusion of sore-throat, and daily notifications of scholars excluded to the Medical Officer of Health, will arise from their affording (1) the possibility of detecting mild or suspicious cases, which would otherwise pass unrecognised, and act as centres of infection; (2) the exclusion from school of these cases, and of other children living in the same house.

It is especially at the commencement of an epidemic, when the prevalence of diphtheria is rapidly rising above the average of preceding years for a similar period, and when also the percentage of cases at school age to total cases is advancing (as in Chelsea in June and July, 1896), that the co-operation of the schools in limiting the spread of the disease is required. From what has gone before, it will be evident that it is especially in June and July in London that Medical Officers of Health must be on their guard against unnotified cases, and that at this season the co-operation of the schools is very essential if headway is to be made against the diffusion of infection. Just at this period of the year, for the reasons before stated, notification by medical practitioners is probably of less value than usual in giving early notice of the occurrence of infection; and yet it is essential that early recognition should be carried out, if epidemic prevalence is to be avoided. In the very early stage of an epidemic, the disease might conceivably be stamped out by the early recognition and isolation of unnotified cases; but later on, as is indicated by the experience of Chelsea, the infection becomes widespread, and even the total closure of the schools is comparatively ineffectual in putting a period to the epidemic.

What then ensues is sufficiently indicated by Chelsea's experience in the latter half of 1896. In these six months the number of notified cases was 294, or 158 per cent. in excess of the average of the latter half of the six years 1890-5. The number of deaths in the six months was 60, equivalent to an annual death-rate of 1.6 per 1,000. The death-rate from diphtheria for the entire year was 1.25 per 1,000, or nearly double the average death-rate of the previous six years. The incidence-rate on the population

for the entire year was 57.2 per 10,000, or 137 per cent. in excess of the average incidence-rate of the previous five years. Is such an occurrence as this unavoidable, our present methods of notification, isolation, and disinfection of premises being apparently incapable of stopping it? Or are there other means which could be adopted, and which hold out a better prospect of success? Whatever be the organisation eventually adopted that would secure the co-operation of the schools in the manner above indicated, it should be made applicable to the whole of London. For the purposes of control of infectious disease, the metropolis must be taken as a whole; and the organisation for this purpose must be capable of application at any time, and at any part of London where it is required.

The only other point which suggests itself for discussion, as regards measures of prevention and the stamping-out of a threatening epidemic, is as to whether urgency could not be declared for the affected district in respect of the removal of diphtheria cases to the hospitals of the Metropolitan Asylums Board. It has unfortunately happened of recent years, and is likely to happen again, that the diphtheria wards of the fever hospitals become full in the summer, and a delay of one or more days is experienced in effecting the removal of patients from their homes to hospital. This delay is especially serious in a district in which diphtheria prevalence is rapidly increasing, and tends to cripple the efforts of the sanitary authorities in other directions to control an advancing epidemic. As I have endeavoured to establish in this paper, the non-removal of patients at times when the disease is quiescent is apparently of much less moment than the failure to do so, when the disease is becoming epidemic, and the activity and propagating power of the infection are at a maximum.

What I would suggest is, that on the receipt by the Metropolitan Asylums Board of a communication from the Medical Officer of Health of a certain district that diphtheria threatened to assume epidemic virulence in that district, confirmed by a statement to a similar effect from the County Medical Officer of Health, the Board should declare urgency for that district, and give directions to its officers to give precedence to applications received from the district in question for removal of diphtheria cases over similar applications from other districts, where urgency had not been declared. It seems to me that some such

arrangement as this is really necessary, unless we are to allow our practical methods to lag behind our increasing knowledge in the science of epidemiology; and that London as a whole would benefit by a greater concentration of the efforts of the central authorities in limiting and circumventing localised outbreaks of diphtheria.

THE SEASONAL FLUCTUATIONS OF EPIDEMIC DISEASES.

By ANDREW DAVIDSON, M.D., F.R.C.P., Edinburgh.

(*Read before the Epidemiological Society, Friday, May* 21, 1897).

A very considerable part of epidemiology is comprised in the study of the multiform fluctuations in prevalence and intensity to which epidemic diseases are subject. One important and interesting chapter in the science has to do with the historical fluctuations of epidemic maladies, but with these we are not at present concerned.

Then we have the secular fluctuations, the causes of which are still involved in the deepest obscurity. Certain diseases, notably scarlet fever and diphtheria, are observed to decrease in prevalence and virulence over comparatively limited regions, and, after remaining quiescent for, it may be, a generation, come to occupy once more the first rank among fatal epidemic maladies. Ransome and Whitelegge have drawn attention to another mysterious class of secular movements, apparently cyclic in character, manifested by small-pox, measles, and whooping-cough, which well deserve closer study.

Perhaps of more practical interest are those fluctuations to which the term "epidemic wave" is generally applied. They may be called (perhaps not very appropriately) *temporal* fluctuations, to distinguish them from the historical and secular movements to which I have just referred. As they influence to a large extent the seasonal fluctuations, it is necessary to distinguish the different movements included in the term, "epidemic wave."

(1) We have the automatic or agglomeration wave, appearing automatically, as it were, every other year in large towns, which is evidently connected with, although not entirely dependent upon, an increase in the number of the susceptible. These waves may be simply the result of an increased prevalence of the disease, or the prevalence may be accompanied by a slightly - augmented case mortality.

(2) There are virulence waves occurring at short but irregular intervals. These seldom extend over more than

a limited area, and last only for a brief period, increasing the fatality of the automatic wave, and modifying the seasonal incidence of a disease in the manner I shall afterwards explain.

(3) There is another class of virulence waves which are pandemic in their range, an example of which is familiar to us in the remarkable extension of small-pox in 1869-72: when the disease in a fatal form swept over India, China, the whole of Europe, North America, the West Indies, extensive regions of Africa, and parts of Polynesia.

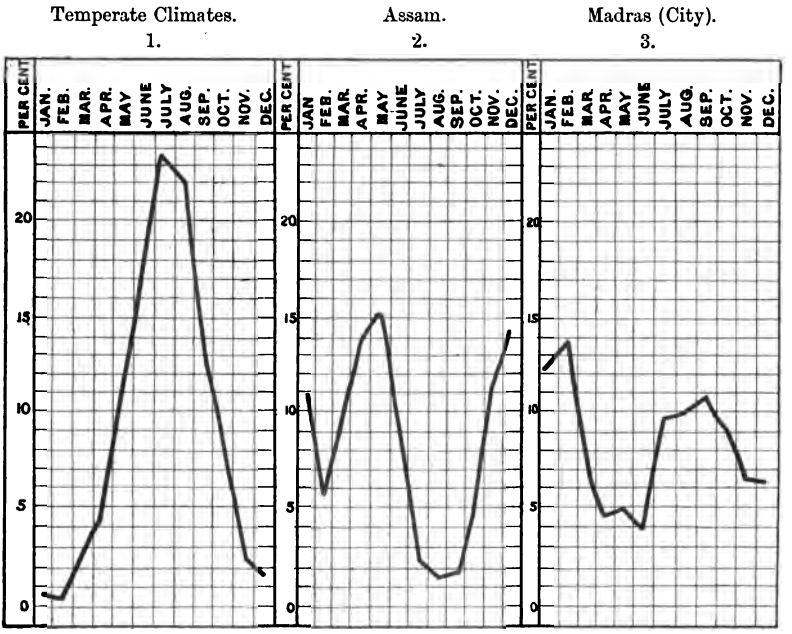
Measles, also, has assumed at times something approaching a pandemic extension; as, for example, in 1846-47, when it prevailed widely in Northern and Western Europe as well as in North America; but neither measles nor scarlet fever has ever attained the world-wide diffusion, or exhibited the exalted intensity, which has characterised small-pox and some other epidemic diseases.

These pandemic waves materially affect the seasonal incidence of a disease.

Finally, there are the seasonal fluctuations of epidemic diseases, with which we are now specially concerned. If we hesitate in subscribing to the view of Sydenham that "a knowledge of the seasons wherein diseases occur is of equal value to the physician in determining their species and in effecting their extirpation", we nevertheless think that a study of these seasonal fluctuations, and of the causes which give rise to them, is not without importance viewed from the standpoint of epidemiology. We cannot hope fully to understand the nature of the other movements and waves to which we have referred until the causes of the seasonal fluctuations are better understood.

Various classifications of epidemic diseases founded on their seasonal prevalence have been suggested. Sydenham believed that all epidemic maladies are referable to two classes, vernal and autumnal. It is needless to say that this classification is an arbitrary one, although it may have been justified to some extent in his own day when malarial diseases occupied such a dominant place in the pathology of England. Some recent epidemiologists have classed epidemic disorders into hot and cold weather diseases. This classification is founded exclusively on considerations of their seasonal prevalence in temperate climates. Diseases occurring in spring may correctly enough be spoken of as cold-weather diseases in northern latitudes, but they are hot-weather maladies in tropical countries, where the maximum temperature is attained in the spring months.

Besides, it must not be forgotten that the seasonal incidence of the same disease often varies in different regions. Asiatic cholera, for example, is eminently a hot-weather disease in temperate climates where the soil generally contains sufficient moisture for its development at all seasons, but where the temperature necessary for the growth of the cholera vibrio is only attained in the summer season. In those provinces of India, on the other hand, where cholera is epidemic, dying out in winter, and in some years altogether absent, the disease does not attain its maximum at the period of the maximum temperature in May or June,



Cholera in Epidemic and Endemic Areas.

but later, when the soil, which has been parched during the winter, has been sufficiently saturated by the rains to permit its epidemic spread. In those provinces of India, again, where cholera is endemic, its seasonal fluctuations differ widely from those observed in temperate climates, or in the epidemic areas to which we have referred. Instead of the single curve we have a double one, a double rise and a double fall dependent on temperature, rainfall, or flooding, on the subsidence of the rains or floods, and on drought.

Diagram 1 illustrates the seasonal oscillations of cholera in temperate climates and epidemic areas; diagrams 2 and

3 show the seasonal fluctuations of the disease in the Surma valley of Assam, and Madras, where it is endemic. The curves for Bengal and Bombay are essentially the same as that of Assam.

In Madras, where the season of rainfall is reversed, the soil sandy, the temperature higher and more uniform than in Bengal and Bombay, and where the drought from February to June is unfavourable for the growth of the comma bacillus, the double curve of endemicity is modified in accordance with the altered conditions of climate and soil. The meaning of these somewhat complicated oscillations of cholera in its endemic haunts will be readily understood, if studied in connection with the climatic factors already mentioned. It will be seen that it is no easy matter to label cholera, in an off-hand way, as a disease peculiar to any particular season, and if we class it among the summer diseases, this classification does not apply to the regions where it is endemic.

The key to the interpretation of the seasonal fluctuations of epidemic diseases must be sought for in the characteristics of the parasites on which they depend, on the influence of climate on their growth and diffusion, or on the susceptibility of the subject to contract the infection. It is difficult to suggest an entirely satisfactory and exhaustive classification of epidemic diseases on the basis of their seasonal prevalence, but three natural groups may be distinguished which include some of the more important maladies.

(1) We have the group of summer diseases, non-contagious in the stricter sense of the term, caused by facultative saprophytes. The seasonal incidence of this group is determined by meteorological conditions peculiar to season, acting on the micro-organism of the disease in the soil or other surroundings of man. Summer cholera, Asiatic cholera in epidemic areas, typhoid fever, yellow fever, and the summer-autumn class of malarial fevers belong to this group.

(2) Then there are the contagious maladies proper, transmissible directly from the sick to a healthy and susceptible subject, such as small-pox and measles, due to purely obligatory parasites, the seasonal fluctuations of which are, I conceive, determined by climatic agencies acting on the human body, rendering it more susceptible to contract the infection at one season than another.

(3) We have another class of diseases, eminently contagious as well as infectious, of which scarlet fever is the most typical example. These are caused by pathogenic

agents reproduced within the body, but which are also capable of growing and multiplying in external media. The seasonal prevalence of this class of maladies may, in one country, be determined by climatic conditions influencing the growth and diffusion of the germ as a saprophyte, in another country by conditions modifying the susceptibility of the body to contract the infection. Hence in one country scarlet fever is seen to prevail in summer and autumn, in another in winter and spring.

The explanation I have offered of the seasonal prevalence of the summer group is, I believe, that which is generally accepted and need not occupy us further. The problem presented by the seasonal fluctuations of scarlet fever is one of much greater difficulty; but it is a problem which cannot be solved, or even hopefully attempted, until the causes of the seasonal evolution of the purely contagious group have been determined. This accomplished, the conception of the relations of the seasonal fluctuations in the prevalence of these three great groups, to the life-history of the infectious agents on which they depend, becomes intelligible, and we will then be in a position to investigate the adequacy or inadequacy of the explanation I have ventured to offer, of the seasonal peculiarities of scarlet fever and its congeners.

The causes of the seasonal evolution of small-pox and measles will therefore occupy our attention to-night. I shall look at the subject in its wider aspects—geographical and historical—rather than with reference to the climatic conditions under which these diseases appear in any particular country.

Small-pox and measles have many epidemic characters in common. They have two distinct seasons of prevalence, namely, late autumn and early winter, and again in spring and early summer. They appear almost simultaneously at the same seasons (the months being reversed) in the northern and southern hemispheres.

It is evident that the difficulty of accounting for the seasonal fluctuations of contagious diseases is much greater than in the case of the malarial and summer groups. That they should have any fixed seasons of prevalence is of itself remarkable. These diseases being caused by obligatory parasites which do not multiply outside the body, cannot be so directly influenced by the varying climatic conditions peculiar to the different seasons of the year as those dependent on saprophytes. If the virus of small-pox or of measles be introduced into a susceptible community,

we know that it often enough spreads, whatever may be the season of the year, or the kind of weather that may happen to be prevailing. Now, as the time when the virus is introduced in any particular instance must be largely a matter of accident, it might have been expected *a priori* that their season of epidemic spread would also be variable. Having once got a start, a contagious malady might have been expected to spread independently of season until all the susceptible had been attacked; or, to use the language of metaphor, that the fire having once been lit, it would burn continuously until all the combustible material within reach had been consumed. This, indeed, is the case as regards a particular locality, a small town or a village, but it does not hold as regards a large city like London, or the country generally. The disease pursues its course in the locality where it has broken out, but it has little tendency to establish new centres, except in winter and spring.

In the case of a purely contagious disease, again, social circumstances peculiar to a nation or locality, especially such as bring together crowds of susceptible subjects in church, school or market, must necessarily have an important influence not only on the actual amount of the disease, but also upon the periods of its increase and decrease. In the same way, the habits of life peculiar to the different seasons, the overcrowding and less perfect ventilation of dwellings in winter, the greater or less facilities or inducements which the seasons may afford for ablutions, and many minor habits of life peculiar to each season, must affect the spread of a contagious disease. As all these social factors, fixed enough as regards time for each country, vary in different regions, one would have been prepared to find correspondingly wide variations in the seasons in which these diseases prevail in different regions. While the spread of these diseases is thus contingent on so many fortuitous factors, nothing is more remarkable than the wonderful regularity with which small-pox and measles observe their stated seasons of increase and decrease in both hemispheres.

The seasonal evolution of measles may be referred to two types, both of which are represented within the limits of our island. The two types are represented in diagrams 4 and 5, showing the monthly distribution of deaths from measles in London, and in the Scotch towns respectively.

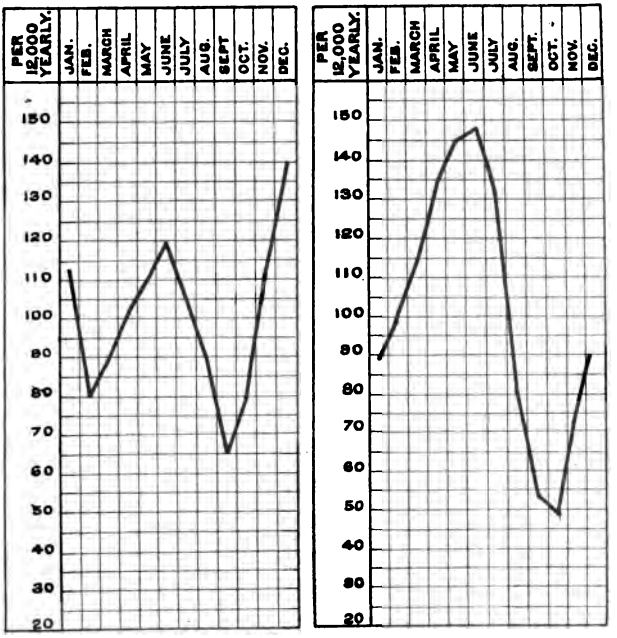
It must be remembered that the death-curve in measles is found to follow very exactly the case-curve, at the

interval of a month, so that in calculating the progress of the case-mortality, the deaths of one month must be credited to the cases of the previous month. The above diagrams represent deaths; the rise and fall of the cases will be a month earlier than is represented in the charts.

The Lancashire towns, and some others, follow more or less the London type; and the ten-year chart published by Littlejohn shows that, of late years at least, the winter rise

London. Scotch Towns.

4. 5.

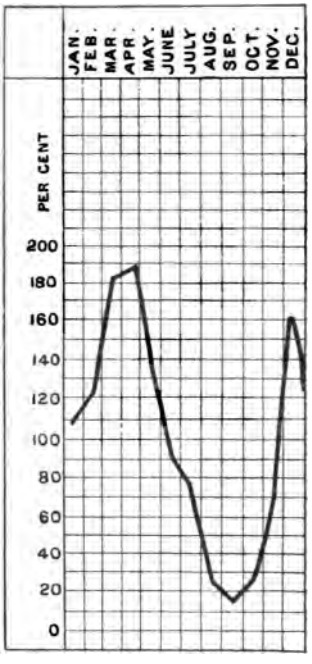


Monthly Distribution of Deaths from Measles.

has been well marked in Edinburgh. The same type is observed in the Netherlands, Hamburg and Scandinavia; in the North-east coast, the inland-mountainous, and the northern-lake regions, of the United States. The Scotch type, on the other hand—in which the winter rise is comparatively slight—is observed in Paris, Berlin, Vienna, St. Petersburg, New York, Philadelphia, and generally, along the middle and South Atlantic and the Gulf and Pacific coasts of the United States. The winter rise tends

almost to disappear in the southern regions where the temperature of the winter months is high. Measles, in India, is mainly a spring-summer disease alike among native and European children.

The only town in the southern hemisphere for which I have statistical data is Melbourne. The disease there has two distinct maximal periods, autumn-winter and spring-summer. From the statement of Scherzer, quoted by Hirsch, it appears that almost all the epidemics observed in Cape



6.—Measles, Edinburgh. (Cases) 10 Years.

Colony have raged from April to June: they consequently here follow the autumn-winter type. Sigaud states in general terms for Brazil, that measles occur in the cold season, at the period when catarrhal fevers prevail; while Bourel-Roncière is only a little more definite when he says that all the great epidemics of small-pox and measles have occurred during the autumn and winter months. We are thus justified in concluding that measles becomes epidemic in winter and spring in the northern and southern hemispheres; its relative prevalence in

winter and spring respectively, varying greatly in different regions.

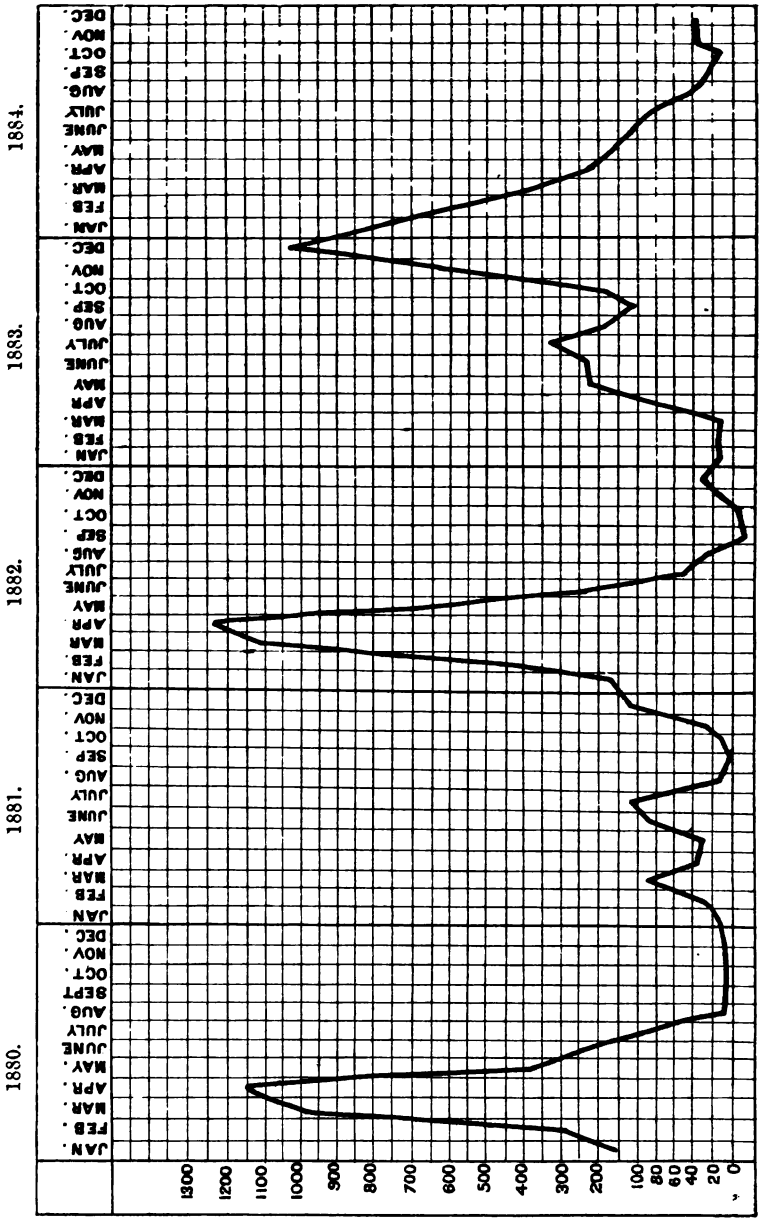
Composite charts are calculated to give an erroneous impression of the manner in which epidemies of measles and small-pox appear and run their course, to those who are not practically familiar with the subject.* On the opposite page is a composite chart (No. 6) representing, according to Herbert Littlejohn, the monthly distribution of measles in Edinburgh for the ten years, 1880-1889. We see the winter rise beginning in October, reaching its maximum in December, falling rapidly in January, then rising slightly in February, to attain its maximum in March and April. It appears from the diagram as if a gradual, uninterrupted, double rise and fall takes place annually. If we glance, however, at the following chart (p. 266), representing five of the years which enter into the composite chart, the mode of its epidemic spread becomes apparent. We observe distinct isolated explosions occurring biennially, confined to the winter or spring months, with sporadic cases and abortive epidemies mostly falling on spring and summer, in the intervening years.

This detailed chart represents not only what occurs in the larger towns, but gives a good idea of how things go in the country generally. More or less isolated explosions occur in particular districts of a city like London, or up and down the country, some in the summer and autumn months, but the great majority in winter and spring. When I examine, for example, the curve of measles in Michigan for 1887, it presents a contour very similar to that of London. For this particular region and year we have a statement of the manner in which this curve is formed. The separate outbreaks of measles in different towns and districts throughout the State in that year numbered 209, which were distributed monthly as follows:—

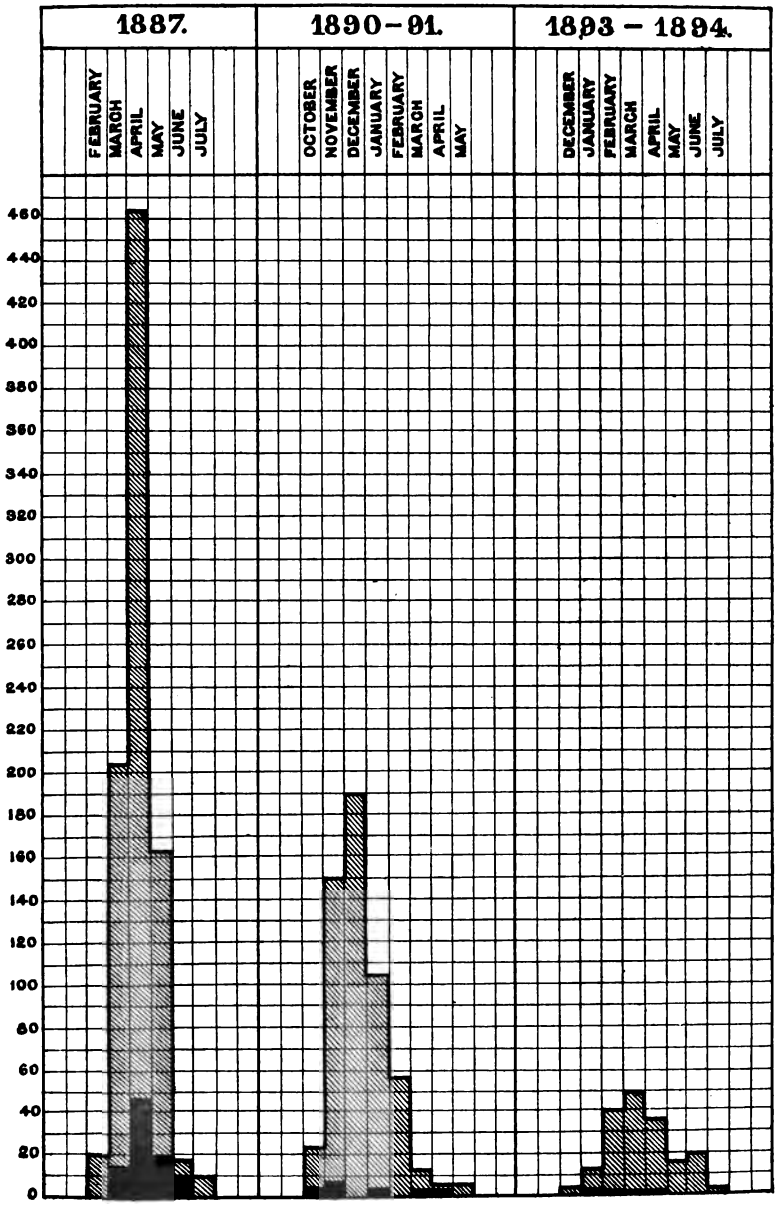
Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
18	11	25	27	28	25	8	3	8	7	21	28

The outbreaks are few in February, just as in London, and again from July to October. The winter rise is in November and December, the spring-summer one from March to June—new centres of infection being more readily set up at these seasons. The same tendency to appear in isolated outbreaks is well shown in charts of epidemies in Gibraltar (p. 267). This feature in the epidemiology both

* By the term *composite* chart I mean one giving the combined monthly or weekly cases or deaths of an epidemic disease for a single locality for a series of years, or for a country or state for one or more years.



7.—Monthly Intimations of Measles (Edinburgh), 1880-84.



8.—Measles Epidemics, Gibraltar (1887-1894).

of measles and small-pox must be clearly realised before the problem we have to deal with can be so much as understood.

From the fact that measles is mainly communicated by direct and close contact, isolated explosions may not be co-extensive with a district, but are apt to be limited to a social class. Frank records an epidemy at Wilna, in 1811-12, which was confined to the Jewish population alone. The explanation of this restriction is to be found in the fact that the children of the Christian population had no dealings with the Jews. It often happens in Germany, where the different classes keep more apart than with us, that an outbreak may rage for a considerable time in a particular social circle before it spreads to another class, living in the same quarter. Measles is thus much more a class disease than small-pox, which strikes at a greater distance.

The biennial wave which we see in the Edinburgh chart, and which is observed in all the large towns, recurs with great regularity. In London it appears for a series of years in succession, then the rhythm is lost for a time to recur again as before. The accumulation of susceptible subjects has doubtless much to do with these biennial explosions, although the breaks in the rhythm, which take place from time to time, seem to indicate that some other factor comes into play. But why does it happen that when accumulation has taken place, and when cases of the disease are present, as they always are in large towns, to start a fresh explosion at any time, that each successive outbreak waits its appointed season of winter or spring to declare itself? It is clear that some special conditions must prevail at these seasons which determine its epidemic spread in these rather than at other periods.

We have shown that the incidence of measles on winter and spring is no *local* one, being observed in all countries. I have to add that it is no *temporary* one, but has been noticed from the earliest times of which we have any record. Rhazes, who first described the disease says, "Care must be taken in the spring and winter of those children who are of a habit of body disposed to the small-pox and measles." His observations were probably made in Baghdad, and I have been furnished, through the kindness of Dr. Sutton of that city, with an official list of the dates of the epidemies of small-pox and measles observed in Baghdad and the neighbouring towns from 1874 to 1895, which I shall here reproduce.

SMALL-POX.

Winter Epidemics.

Began.		Ceased.
1874. Nov. 1st	...	March 6th.
1882. Dec. 15th	...	March 10th.
1884. Nov. 15th	...	Jan. 26th.
1891. Dec. 3rd	...	March 18th.
1892. Oct. 15th	...	March 2nd.
1893. Dec. 20th	...	April 20th.
1894. Oct. 15th		

Spring Epidemics.

1878. April 8th	...	June 18th.
1883. March 5th	...	June 12th.
1886. March 5th	...	June 1st.
1889. March 3rd	...	April 26th.
1891. April 2nd	...	Beginning of June.
1895. March 15th	...	May 3rd.

MEASLES.

Winter Epidemics.

Began.		Ceased.
1874. Nov. 1st	...	Dec, 22nd.
1888. Sept. 15th.	...	April 26th.

Spring Epidemics.

1879. March 2nd	...	June 8th.
1883. March 5th	...	May 20th.
1884. April 6th	...	June 2nd.
1886. March 5th	...	May 2nd.
1888. April 5th	...	May 18th.
1892. May 8th	...	June 26th,
1893. March 15th	...	April 24th.

This table shows that, from the time of Rhazes to the present day, small-pox and measles appear regularly in winter and spring in Mesopotamia. The frequency with which both diseases are seen to break out from the 2nd to the 5th of March, and their entire absence during the summer months are remarkable.

It may be as well to mention in passing that, from June to October, when measles and small-pox are absent from Baghdad, the weather is absolutely rainless and excessively hot. The same is the character of the summer months in Gibraltar, and in other places, where these diseases disappear at that season. This has to be kept in mind in connection with the theory that the setting in of the rains in India is the cause of the subsidence of small-pox.

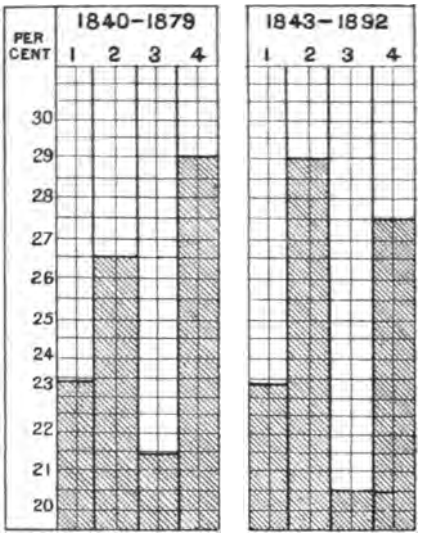
As in Baghdad, so in Europe and America, measles has been a disease of winter and spring from the earliest times. In the seventeenth and eighteenth centuries it occurred in London, just as it does now, in winter and spring outbreaks; the winter ones being perhaps less severe than at the present day.

A few other points respecting the winter and spring epidemics deserve notice. We find from the Registrar-General's annual summaries that the quarterly mortality from measles in London has changed within quite recent years. Referring to the annual summary for 1880, which gives the weekly average of deaths for each quarter for the forty years, 1840-79, and that for 1892 giving the average for the fifty years, 1843-92, we find that the quarterly distribution of deaths has altered in the manner indicated in the following diagram (No. 9):—

In the first series of years the maximum mortality falls on the fourth quarter, in the second series on the second quarter. This change in the quarterly distribution of measles is not an accidental one, but is related to an increased fatality of the disease from 1881 onwards.

That the change from the winter to the spring type arises from an increased prevalence and fatality of the disease, will be evident if we throw into the form of a diagram the quarterly distribution per cent. of the deaths in London in what we may call epidemic and non-epidemic years (Diagram No. 10).

In epidemic years the force of the disease is seen to fall



9.—Quarterly Distribution of Measles Deaths (London).

on the second quarter, in non-epidemic years on the fourth quarter. The rise in the second quarter is not the result merely of an increased prevalence of the disease, but the case-mortality is also higher. In Hamburg at least, the case-mortality in the winter outbreaks for the sixteen years, 1879-1894, was 4.14; in spring, 5.10 per cent. As the force of the severer outbreaks, caused by virulence waves, falls on spring, this will account for the higher case-mortality at this season. Whether, apart from the intrusion of these waves, the case-mortality is higher in spring than in winter, is doubtful.

The virulence of measles, as estimated by the case-

mortality, bears a very constant relation to the annual course of the disease, as will be seen from the following figures of the cases, deaths, and percentages of deaths to cases, in thirteen four-weekly periods deduced from the Hamburg returns for the sixteen years, 1879-94. The deaths are credited to the cases of the previous month, for the reason already given.

Four-Weekly Periods.

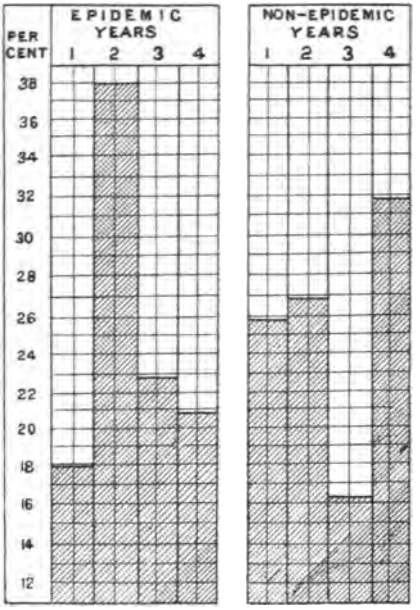
	1.	2.	3.	4.	5.	6.	7.
Cases	4,017	3,568	3,525	3,564	6,888	10,721	9,284
Deaths	166	136	180	255	444	508	256
Percentages	4.1	3.9	5.1	7.2	6.4	4.7	2.8

	8.	9.	10.	11.	12.	13.
Cases	4,695	2,382	2,637	4,094	5,638	5,359
Deaths	166	101	156	205	213	167
Percentages	3.5	4.2	5.9	5.0	3.8	3.1

During the third period, when the preceding winter outbreak has exhausted itself, and when the cases for this season have reached their minimum, the case-mortality has already begun to rise. The spring rise in prevalence begins in the fourth period, but is still very slight; but during this early extension there is a further increase in the case-mortality. With the first epidemic bound, however, in the fifth period, corresponding to the end of April and beginning of May—when the cases are nearly doubled—the case-mortality begins to fall; so that when the maximum prevalence is attained in the sixth period, a notable decrease in the fatality of the disease is observed, which becomes still more pronounced in the seventh period, when the disease, although still wide-spread, is beginning to decline. As soon, however, as the first decided fall in the number of cases takes place in the eighth and ninth periods, the case-mortality begins to rise, and continues to do so until the eleventh period, when the winter extension has made progress; the disease then becomes less fatal as it becomes more prevalent. It seems as if the virulence of the disease increased during the inter-epidemic periods when the cases are at their minimum, and that it still further increases during the first slight extensions of the epidemy, and then decreases as the disease becomes general, sweeping all the unprotected into its net.

I take it, that only the more susceptible are attacked when the disease is slightly diffused. In passing through these slightly resistant subjects, the virus may be supposed to gain in virulence. This virulence is further increased during the fourth period coincidently with the first slight

extension, when, it may be assumed, the very susceptible are still being selected. The increase of virulence will favour the diffusion of the disease; but, as the disease becomes widespread, and is consequently brought home to all, the less susceptible are attacked, and the case-mortality diminishes, either because the persons now attacked are better able to resist the malady and so recover in greater proportions, or because, in passing through less receptive subjects, the virus becomes attenuated This is a theory; but the increase of the case-



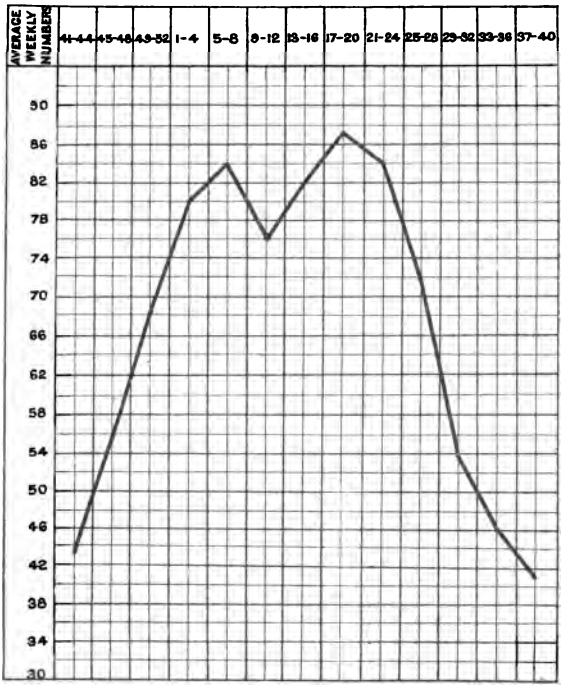
10.—Quarterly Distribution of Measles Deaths (London).

mortality from the third to the fourth period, when the disease is beginning to spread, cannot easily be explained on any other hypothesis than that of an actual increase in virulence. I have been somewhat minute in describing the evolution of measles in respect to prevalence and virulence in Hamburg, because I think it represents the normal course of measles in its annual evolution in other countries, and also because small-pox, so far as the data at my disposal indicate, follows pretty much the same course.

The increase and decrease of virulence during the inter-epidemic and epidemic periods may be ascribed to an

attenuation or exaltation of the virus according to the greater or lesser susceptibility of the subjects attacked; but why the epidemic periods should everywhere fall on winter and spring rather than on any other season, is a question to which the natural evolution of the disease affords no answer.

Small-pox, like measles, is an autumn-winter and spring-summer disease, as will be seen from the following charts



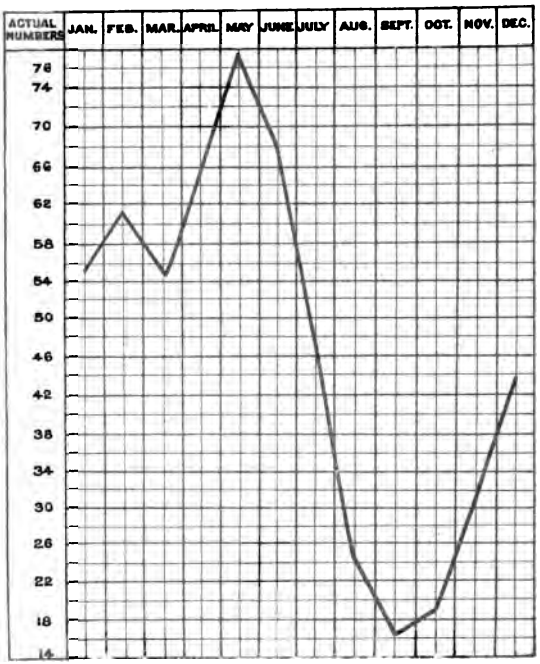
11.—Small-pox (London) 50 Years' Average. 13 Four-Weekly Periods.

representing its monthly distribution in London, New York, Madras, Bengal, and the Punjab.

The winter rise of small-pox commences in October in London and New York, and in November in Bengal, the North-West Provinces, the Central Provinces, the Hyderabad Assigned Districts, Bombay, and Burma. In Madras it appears a month later. In all these regions, with the exception of Madras, the winter rise corresponds to the first notable fall in the temperature. It is deserving of remark that in London small-pox and measles begin to

increase in prevalence in the fortieth week, and bronchitis in the forty-first week. These coincidences in point of time cannot be without significance in relation to the causation of their seasonal spread. The period of the subsidence of the winter outbreaks, as well as the time when the spring rise attains its maximum, is more variable. Small-pox, especially when it assumes a severe type, tends to be protracted into summer.

The amplitude of the small-pox curve does not follow very closely that of the temperature; nevertheless, where

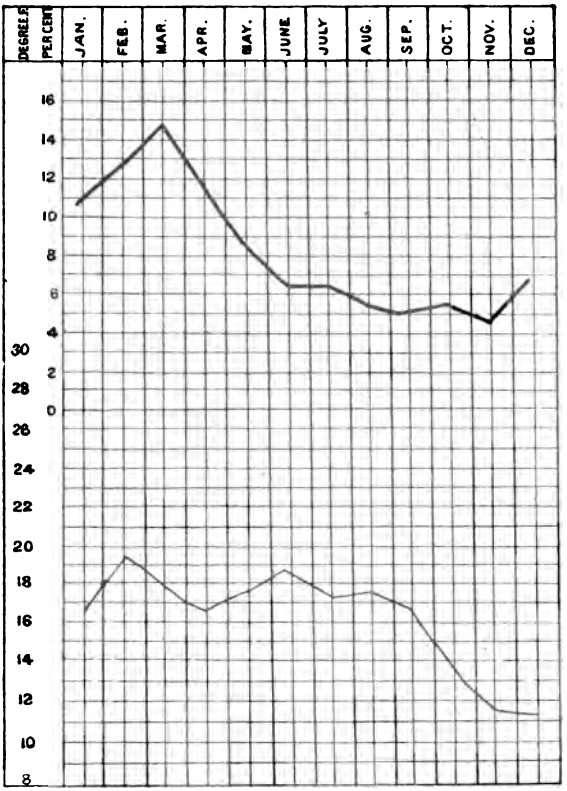


12.—Small-pox (New York), 1871-1887.

the temperature of the year is comparatively uniform, as in Madras, the seasonal fluctuations of small-pox are also less pronounced. The spring rise appears, upon the whole, to be most marked in those regions where the ranges of temperature are greatest; the winter rise comes into prominence only in those parts of India, such as the Punjab, where there is a distinct winter season. Some correspondence is also to be observed between the periods of the small-pox and range maxima. Thus, in Bengal, where the mean daily ranges of temperature begin to

decline from April to May, the maximum of small-pox is attained as early as April; whereas, in the Punjab, the maximum, both of small-pox and of the temperature range, occurs in May.

Small-pox, like measles, shifts its incidence in epidemic years from the fourth and first to the second quarter, as will be seen from diagrams 16 and 17, representing the

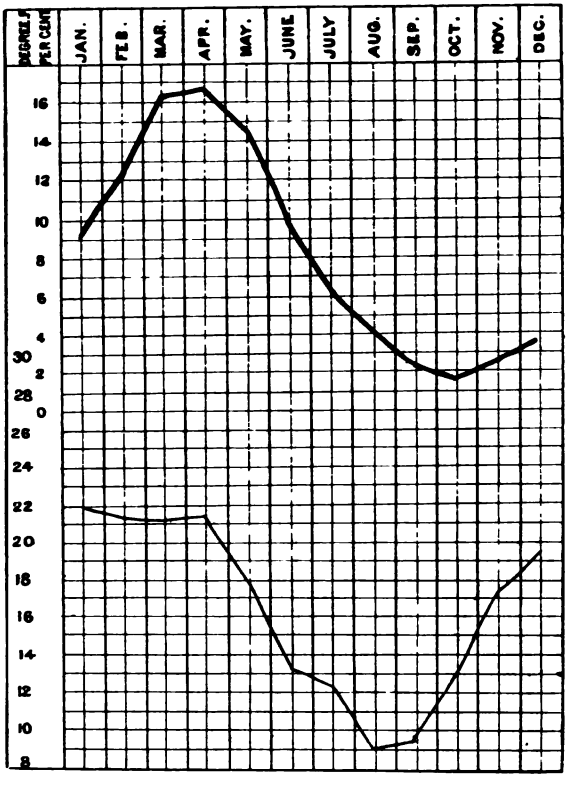


13.—Small-pox and Daily Range (Madras).

quarterly distribution of deaths from small-pox in London and the Punjab, in epidemic and non-epidemic years.

This shifting of the mortality to spring in epidemic years, so noticeable in measles, is clearly the result of the intrusion of virulence waves into the seasonal sphere. It indicates a disturbance of the normal evolution of these diseases, and is not dependent, I think, on the causes that give rise to the seasonal fluctuations.

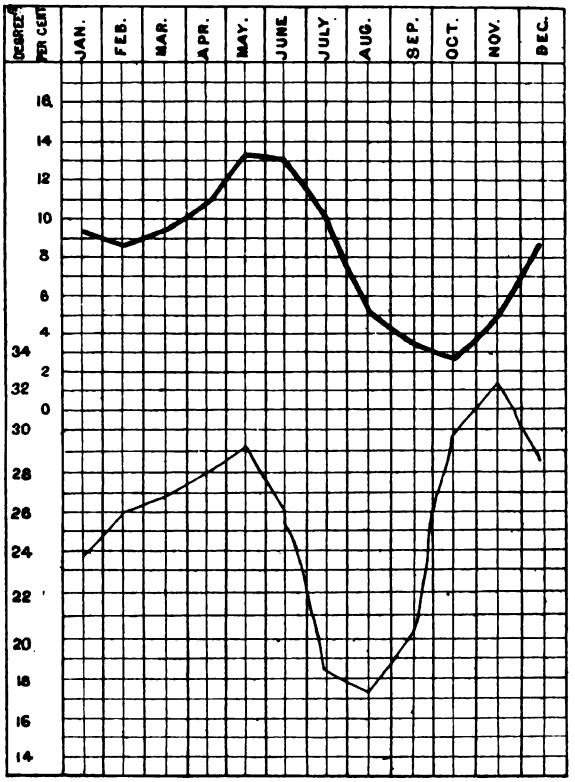
The present seasonal distribution of small-pox in England and other civilised countries is modified by vaccination, which, by rendering epidemics less frequent and severe, depresses the spring rise. This will be seen by comparing the curve of small-pox, deduced from a fifty years' average, with that of the severe outbreak of 1871-72, when it temporarily assumed something approaching its normal epidemic type.



14.—Small-pox and Daily Range (Bengal).

From the remarks of Rhazes, already quoted, we know that small-pox has been a disease of winter and spring from the earliest times, but the precise period when the spring outbreaks have attained their maximum has varied considerably at different epochs in England: the period of the spring rise has not appreciably fluctuated. When associated—as it often was in the seventeenth century—with spotted fevers, small-pox frequently prolonged its

reign well into autumn, the maximum being in many years attained as late as July and August. The bills of mortality prove that Sydenham was speaking from observation when he classed small-pox, in years when "it had the ascendency over other diseases", along with plague as a disease "which begins in spring, increases daily, and reaches its climax about the autumnal equinox". Winter

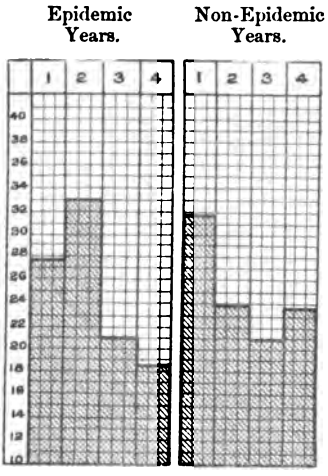


15. Small-pox and Daily Range (Punjab).

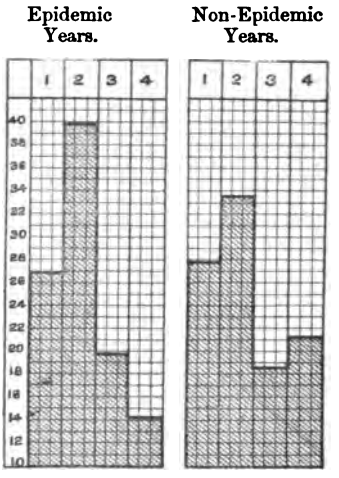
epidemies of small-pox, however, were not unknown in the seventeenth century. Small-pox has generally been observed to assume a severe or even malignant type when typhus has been prevalent. Rutty, for example, writing of the typhus in Dublin in 1758, says: "As the cases of petechial fever increased much beyond the usual number in January, it was observable that the small-pox kept pace in malignity with the fever": this is an example of a winter outbreak

being protracted by association with typhus. I do not think we shall be far wrong in ascribing the prolongation of the small-pox into summer and autumn in the seventeenth century to its unusually severe type caused by its association with spotted fevers.

The regularity with which small-pox and measles everywhere, and in all ages, become epidemic in winter and spring, forbids us referring the phenomenon to accident. Their greater prevalence at these times must be the result of some mysterious evolutionary property inherent in the germs of these diseases, or must be indirectly or directly dependent on season, or, rather, on certain climatic conditions peculiar to season.



16.—Quarterly Incidence of Small-pox (London).



17.—Quarterly Incidence of Small-pox (Punjab).

How can we imagine an evolutionary property of a germ which shall determine its activity in a given locality in one year in winter and in another year in spring? The irregular way in which these explosions occur cannot be reconciled with the evolutionary hypothesis.

Flügge, the distinguished bacteriologist, adopts the view that the relation of small-pox to season is an indirect one. "One can scarcely go wrong", he says, "in attributing the regular increase of small-pox in winter to the greater amount of time spent at home, to the more intimate association of individuals, to the wearing of the same clothing for some time, and to the diminished cleansing of the skin: thus rendering the transport of the infective agents and

their preservation at the seats of invasion much easier than in summer, when all these favouring factors are absent." "These differences", he continues, "are of course more or less marked in different countries, according to the climate and mode of life. In tropical climates, the remarkable diminution of small-pox in the rainy season may be due to the easier cleansing of the body and to the increased difficulty in the transport of the infective agents by currents of air; perhaps also to certain habits of life which cannot be thoroughly appreciated." The factors here invoked are too multiform and variable to furnish a satisfactory explanation of a singularly uniform phenomenon. The spring rise and summer prevalence of the disease are ignored, as is the fact that in a particular locality, the disease does not begin in winter and continue on through spring into summer, but falls, as we have seen, in one year in winter and in another year in spring. The remarks about winter answer well enough to the circumstances of life in the latitude of Britain or Germany, but are quite inapplicable to India or to the tropics generally. If the setting-in of the rains arrests the progress of small-pox in India, what, we may ask, arrests it in Mesopotamia, and the numerous other regions, where the summer is rainless?

That the influence of climate on the seasonal recrudescences of these diseases is a direct one may be inferred from their occurrence at corresponding seasons in the northern and southern hemispheres; from the winter rise being to a marked degree proportional to the fall in the temperature, and from its appearing in the month when the first notable fall occurs; from the relation of the spring rise to the temperature ranges; and, finally, from the winter and spring recrudescences appearing along with, and following closely the march of, other maladies, the seasonal evolution of which is known to be largely determined by climatic conditions.

Assuming, then, that the winter and spring prevalence of small-pox and measles is due to some climatic factor or factors peculiar to these seasons and not to social habits, it will facilitate their identification and their mode of operation, if we can decide in advance whether the climatic conditions which determine the seasonal evolution of small-pox and measles do so by their action on the pathogenic agents of these diseases outside the human body, or by rendering the body itself more susceptible to the infection.

Light, heat, humidity and other weather conditions have

been thought to act directly on the virus of small-pox and measles by increasing or diminishing its amount, vitality, or infectiveness, or by favouring or inhibiting its diffusion.

There is no difficulty in understanding the direct action of climatic agencies on facultative saprophytes, and the manner in which they regulate their seasonal prevalence. The growth of these organisms is directly influenced by the temperature and humidity of the media in which they grow. The case is different in respect to obligatory parasites. Their soil is the human body. There is no evidence that climatic conditions affect their multiplication or modify their infectiveness. Hence, there is a marked distinction between diseases caused by saprophytes and those due to obligatory parasites in respect to their seasonal and geographical distribution. The former do not become widely epidemic in regions where the summer temperature is low, nor in temperate climates except in summer, nor in the tropics if the necessary humidity is wanting. If small-pox and measles become epidemic in winter and spring, it is not because an *optimum* temperature or humidity is then attained, for the winter and spring temperatures and the winter and spring humidities at which they spread vary extremely in different countries, and in the same country in the winter and summer seasons. Nothing, indeed, is more remarkable in the epidemiology of these maladies than the extreme differences in the meteorological conditions under which they become epidemic. I witnessed a destructive epidemy of small-pox in Madagascar in 1868, when the mean temperature fluctuated between 90° F. and 100° F.; in 1870, the disease was observed to spread in the Hudson Bay Territories with the thermometer standing at 40° below the zero of Fahrenheit. This, of itself, appears to be a proof that the seasonal fluctuations in the prevalence of small-pox cannot be ascribed to the direct action of temperature on the infectiveness of its virus.

The opinion has been expressed that epidemies of measles are checked by a rise in the temperature above 60° F., or by a fall below 42° F. These limits have been deduced from observations made in England, and have reference to this country alone. I do not question the accuracy of the statement so far as England is concerned, but other limits would have to be assigned for the Northern States of America or the Northern Governments of Russia, and quite different ones for the Tropics. As a fact, measles and small-pox recognise no *optimum* of temperature or humidity,

but prevail epidemically in winter or in spring, as the case may be, whatever may be the meteorological conditions of a given country at these seasons. This could not be the case if the seasonal recrudescences of these diseases were determined by the action of meteorological conditions on the infectiveness of the virus outside the body.

The aerial diffusion of measles is confined within so narrow limits, that varying facilities for diffusion arising from the kind of weather peculiar to the seasons, cannot be supposed to regulate the periods of its epidemic spread.

The case is somewhat different as regards small-pox. Its virus is capable of being diffused for considerable distances through the air, and this mode of infection must necessarily be favoured, or hindered, by states of the atmosphere. The dryness of the air, and the strength of the air currents, have probably most to do with its diffusion. But these climatic factors are just those, of all others, which are most variable in different seasons and localities. In England, the air is nearly saturated when the winter rise of small-pox and measles takes place; the humidity is at its minimum during the spring rise.

These considerations forbid us to ascribe the seasonal recrudescences of small-pox and measles to the action of climatic agencies on the virus outside the body or on its diffusion. We are thus led by a process of exclusion to the inference that climate determines the seasonal prevalence of these diseases, by rendering the body more susceptible to contract the infection in winter and spring than at other seasons. This conclusion is, I believe, the right one.

It may be remarked that the term "susceptibility" is used to denote: 1. Simple vulnerability of the surfaces through which infection takes, which in this case is the respiratory mucous membrane. 2. A lessened refractoriness of the system to infection. The one is a local, the other is a general susceptibility. Local vulnerability probably plays the most important, although not an exclusive, part in connection with the infection of small-pox and measles. We may err, however, in taking a too mechanical view of the *rôle* of vulnerability of the respiratory mucous surfaces in relation to infection. If the respiratory mucous membrane serves only as the port of entrance for the infective agent, vulnerability will mean nothing more than a lessened power of resistance to the attack of pathogenic organisms; but if it serves also as the seat

of their incubation, the degree and nature of the lesion may exercise an influence on the virulence of the germs, and consequently on their spreading power.

Returning from this digression, I shall now state as briefly as possible my reasons for believing that the susceptibility to contract small-pox and measles is more widely diffused in a community in winter and spring than at other seasons. The proof that this is the case must be to a large extent inferential—not demonstrative. The experimental investigations hitherto made into the conditions favouring infection, unfortunately, do not help us much. Charrin and others have shown that cold diminishes the resistance of animals to many pathogenic agents, while Bouchard's experiments have led him to the conclusion that cold favours the invasion of the system by microbes living on the surface of the mucous membranes. These experiments may be taken to indicate that climatic conditions which lower the vitality of the system generally, or that of the mucous membranes, increase the susceptibility to infection.

Of much greater value for our purpose are the observations which have been made on the connection between fluctuations of temperature and the rise and fall of measles in particular localities. Investigations of this kind are exceedingly laborious, and to be of value must be carried over a number of years: hence, the available material is scanty. Ransome concluded from his observations that the chief undulations of the measles curve rose with the fall of the temperature and *vice versâ*. My own observations are to the same effect.* If this relation is the normal one —for it is impossible in the case of an epidemic disease like measles that it should be a constant one—and if the rise in the deaths indicate an increase in the cases, the only way of accounting for a fall in the temperature being accompanied by an increased prevalence of measles is on

* The lecturer here exhibited a chart showing the remarkable correspondence between the depressions of the mean temperature and the rise in the reported cases of measles in New York, from October 1896 to Feb. 20th, 1897, the cases being credited to the temperatures a fortnight earlier. The figures from which the chart was constructed are as follows :—

		Oct.				Nov.				Dec.		
1896 ...	17	24	31	...	7	14	21	28	...	5	12	19	26
Mean Temp.	53.5	51.3	56.4	...	56.7	46.9	49.1	51.8	...	32.5	45.6	35.3	26.3
Cases ...	53	57	67	...	65	92	86	130	...	152	121	131	160

				Jan.				Feb.		
1897	...	2	9	16	23	30	...	6	13	20
Mean Temp.	...	33.7	37.4	31.4	34.9	19.1	...	29.7	32.3	36.7
Cases	...	165	116	184	164	174	...	143	133	153

the supposition that the cold increases the susceptibility of the community to the infection.

A general survey of the climatic conditions under which the winter and spring recrudescences of small-pox and measles take place in different parts of the world, and a consideration of the manner in which they are seasonally associated with respiratory diseases, point to the same conclusion. Both small-pox and measles, as we have seen, begin to increase all over the world in the month when the first decided fall in the temperature occurs. There are very few, if any, exceptions to this rule. In tropical countries, the winter temperature is high compared to that of northern latitudes, but the transition from the heat of summer and autumn to the comparative cold of winter produces a marked impression on the constitution, manifested in a complete change in the prevailing type of disease which takes place at this season. We are not, therefore, to suppose that the transition from autumn to winter in tropical countries is an inadequate explanation of the comparatively slight increase in the prevalence of small-pox which uniformly appears in the beginning of winter in these regions. Here, then, we have a climatic change so regular in the time of its occurrence, so universal in its extent, and so marked in its pathological consequences, as to account for the almost simultaneous increase in the prevalence of measles and small-pox in all parts of each hemisphere. One has only to compare the very distinct winter rise of small-pox in the Punjab, where the winter fall in the temperature is considerable, with the slight rise observed in Bengal and Madras, to feel assured that the fall in the temperature is causally related to the increase of the disease. A close relation is also observed between the extent of the winter fall of the temperature and the height of the winter rise of measles. It is not, however, so much the actual temperature of winter that is of moment, as the extent and suddenness of the fall.

It cannot for a moment be supposed that this increase in the prevalence of small-pox and measles, in proportion as the contrast between the summer and winter climates of a locality becomes more marked, is the result either of an increase in the amount or in the infectiveness of the virus, or of the greater facilities for its diffusion in cold weather. It can only be referred to an increased vulnerability of the respiratory mucous membranes, or to an exalted susceptibility of the system generally, resulting from the upset

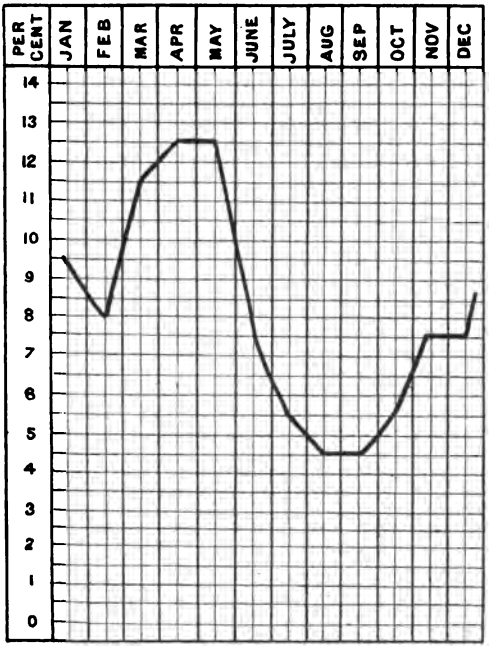
of the physiological balance caused by the transition from the hot to the cold season.

The fall in the temperature in the end of autumn is the most important and obvious factor concerned in this disturbance of the physiological functions which finds its expression in the new train of pathological phenomena which everywhere appears at this season. But the fall in the temperature is correlated to other meteorological factors, such as fluctuations of temperature, atmospheric humidity, fogs, the force and direction of the winds. These subsidiary factors vary in different localities. Hence the height of the winter rise is also seen to vary, being upon the whole more marked in localities where one or more of these factors accentuate the effects of the fall of the temperature. Fogs and humidity are probably largely responsible for the severity of the winter outbreaks of measles in London.

The almost simultaneous rise in the prevalence of measles and small-pox and that of respiratory diseases, and the way in which these two classes of maladies keep step in their annual fluctuations, cannot, as I have said, be without significance as regards the causes of their seasonal prevalence.

Here is a chart (No. 18), after Jürgensen, of the average monthly admissions for pneumonia to the General Hospital in Vienna. It will be observed how closely this curve resembles that of small-pox in various countries. The winter rise of both diseases occurs in the same month as in London; they fall together from January to February; the spring rise in March continued on to May is the same in both diseases. Nor does this chart differ much from that of measles in Edinburgh. If one did not know beforehand that it represented pneumonia, it might be taken as a typical curve of small-pox or measles. The remarkable similarity of the curves can only be explained on the hypothesis that the seasonal evolution of these three diseases is largely determined by the same climatic factors acting on the system in the same way. Now, in what way are the winter and spring recrudescences of respiratory diseases caused? It cannot, I think, be doubted that an increased vulnerability of the air passages determines to a large extent the seasonal prevalence of bronchitis, pneumonia, and sore throat. Under the influences of the fall of temperature and other subsidiary factors, the respiratory tract becomes, for the time being, *the locus resistentiæ minoris;* it is less able to resist the attacks of the pathogenic agents that invade the system

through the air passages. I do not affirm that the increase of these diseases at this season is solely due to a greater vulnerability of the respiratory mucous membrane, but this factor undoubtedly plays an important part in causing this increase. This is evident from their comparative rarity in winter in countries where the winter season is mild; their greater prevalence in spring in continental climates, where the ranges of temperature are extreme—winter and summer being often represented within the space of twenty-four hours—and by their frequency among those whose occupa-



18.—Monthly Admissions for Pneumonia (Vienna).

tions compel them to inhale an atmosphere laden with irritating particles that inflame the mucous membranes. No reason could be assigned why differences in climate, still less why special occupations, should lead to an increase of the microbes on which these diseases are believed to depend, but it is not difficult to understand in what way climate and employment may determine varying susceptibilities to infection. As the infection of measles and small-pox takes place through the air-passages, the increased vulnerability which determines the greater prevalence of

respiratory diseases in winter and spring, furnishes an adequate explanation of the epidemic spread of the former class of maladies at these seasons.

Hebra expresses the experience of the profession when he reckons inflammatory affections of the respiratory organs, influenza and whooping-cough, as precursors of outbreaks of measles. These are diseases that manifestly increase the vulnerability of the respiratory mucosa, from which we may safely infer that a congested, irritable, or inflamed state of the air passages renders the system more liable to contract the infection.

As the winter advances the system gradually adapts itself to the new conditions. It becomes set, as it were, to cold. Hence respiratory diseases, and also small-pox and measles, decline in frequency in January and February. But this decrease is not a permanent one. Other climatic influences now come into play, which give rise to the spring recrudescence of all these diseases. I am inclined to look upon the great ranges and sudden fluctuations of the temperature in spring as the chief, but not the only, factors in determining the spring rise. I have already noticed the relation between extreme ranges of temperature in India and the prevalence of small-pox, and the correspondence in the time when the maximum ranges are attained and that when small-pox reaches its maximum. Having trespassed so much on your time and patience, I shall not enter further into the subject of the spring rise. The same line of argument by which we have tried to connect the winter rise with the fall of temperature, and by which the fall in temperature is inferred to act by increasing the susceptibility of the body to infection, applies to the spring rise as determined by extreme ranges of temperature. I wish, however, before closing, to draw attention to the significance of the following figures of the admissions for tonsillitis and sore-throat in the European army of India for the years 1893-1894.

Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
351	286	351	453	697	430	277	250	244	331	321	356

I judge from these figures that, in India, a special vulnerability of the upper air passages is developed from October to January, and again from March to May. These are the periods when small pox is also most prevalent. The inference that the causes which determine the seasonal fluctuations of sore-throat determine those of small-pox is almost irresistible.

THE JENNER MEMORIAL MEDAL.

At the time of the Jenner Centenary celebration in 1896, it was felt that some action should be taken by the Society—with whose name the subject of vaccination will always be associated—which, while celebrating an event of such importance and of such far-reaching effect to preventive medicine, should at the same time do something to promote the study of epidemiology in the future. It was therefore decided that the Society should found a memorial medal, which should be awarded from time to time for work of great merit; and it was thought that in founding a memorial of this nature the medical profession should be afforded an opportunity of contributing to the fund. It is therefore a matter of much congratulation to be able to announce that the appeal which was made to the profession through the medium of the medical journals has enabled the dies for the medal to be executed at a merely nominal cost to the Society itself, and the fact that the fund was contributed to from many parts of the world cannot fail to enhance the value of the medal in the eyes of its recipients. A sub-committee, consisting of the President, Mr. Shirley Murphy; Sir Richard Thorne Thorne, K.C.B., F.R.S.; Fleet-Surgeon Theodore Preston, R.N.; Dr. Frank Clemow; Dr. Sweeting, the Hon. Treasurer; and Drs. Bulstrode and Coupland, the Hon. Secretaries, was appointed to consider the designs for the medal, and this sub-committee was, upon the retirement of Mr. Shirley Murphy from the presidential chair, still further strengthened by the advice of Prof. Lane Notter, the succeeding President. It was eventually decided to recommend the council to accept a design submitted by Mr. Allan Wyon, F.S.A., and its execution is now in his hands. It is hoped that in the next volume of the *Transactions* a facsimile of the design for the medal will be presented.

The following are the names of the subscribers to the fund:—

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OBITUARY NOTICE.

Sir Benjamin Ward Richardson, M.D., F.R.S., Etc.

Although the late Sir Benjamin Richardson devoted only a small part of the multifarous activity of his life to the actual study of epidemics, yet his work as a sanitary reformer, and his constant advocacy of measures which he deemed calculated to improve the public health, constitute a distinct claim upon our pages.

Benjamin Ward Richardson was born at Somerby, in Leicestershire, in 1828, and the last completed act of his life was to revise the concluding chapter of a volume of "Memorials," from which much interesting information about the author may be gleaned, although unfortunately the book is but imperfectly supplied with dates, even in relation to the principal events of his life. It appears that his mother, who died in his early childhood, had not only destined him for the medical profession, and sketched out the road by which he was to attain to it, but that she had also succeeded in impressing upon him the value and the importance of the work which he would be called upon to perform. His father, who lived to a great age, was anxious to carry the maternal wishes into effect; and hence the whole training of the future physician was so directed as to have some bearing upon his intended profession. He was first sent to a dame school, presumably in his native village; and, after a short time at a preparatory school of a more advanced character, he went to another, at which he remained for some years, and of which we are only told that it was in Leicestershire, and that it was presided over by the Rev. W. Young Nutt. Of this school young Richardson seems to have retained none but pleasant memories; although his account of his pursuits there would tend to suggest that he must have been what most schoolboys would call a "sap." On leaving it, he was apprenticed, according to the fashion of those days, to Mr. Henry Hudson, a medical practitioner at Somerby, and was there initiated into the routine duties of country medical life, with the result that he became familiar with all kinds of professional duty and emergency, and ready to turn his hand to anything which might be required of

him. The days of apprenticeship are gone beyond recall; but it is impossible not to feel that, in many respects, they conferred great advantages upon young men who were fortunate in the masters with whom they were placed; and Richardson, from his own account, seems to have been thus favourably circumstanced. From Mr. Hudson he went to study at what was then called "Anderson's University" in Glasgow, and from there, as a sort of unqualified assistant, to a brother of Mr. Hudson's, at Saffron Walden. Ultimately, he passed the qualifying examination of the Faculty of Physicians and Surgeons of Glasgow, and accepted an invitation to go to Mortlake, in order to assist, and to conduct a branch practice for, Dr. Willis of Barnes, a scholarly and highly-accomplished physician, who wished to relieve himself of the drudgery of attendance on some of the outlying portions of his district. In 1853, while Richardson was still at Mortlake, the writer of these lines first made his acquaintance, and laid the foundation of a friendship which, in spite of differences of opinion on many subjects, remained undisturbed to the last. At Mortlake he was indefatigable, alike in the treatment of the sick, in the investigation of disease, and in his communications, always interesting and often highly original, to the British Medical Association and other Societies. On the appearance of cholera in England in 1853, he succeeeded in founding among his medical neighbours the "East Surrey Society for the Investigation of Cholera", which held its meetings in the Hawkstone Hall in the Waterloo Road, and in which the writer succeeded him as Honorary Secretary in 1854, when Richardson had obtained the degree of M.D. from the University of St. Andrews, and had decided to try his fortune in London. He settled in Hinde Street, next door to the house in Manchester Square in which he died; and having converted his coach-house into a laboratory, continued for many years to devote much of his time, and practically the whole of his leisure, to physiological and pathological research. As the first fruit of his labours in these directions he obtained, in 1856, the Astley Cooper Triennial Prize of £300, for the best essay on the Coagulation of the Blood; and, in relation to this, he was the first to point out that some of the conclusions which he had maintained required to be re-considered by the light of fresh evidence. Soon afterwards he published a paper on fibrinous deposits in the heart, as one consequence of which he soon began to take definite rank as a consultant. Previously to this, he had either had no patients in London, or, as is

most likely, none of a remunerative kind; and it is said that when, soon after the publication of his paper, a gentleman went to seek his advice, Richardson, quite unprepared for such an occurrence, came into the room in a green-baize apron, with a file in one hand, and a piece of brass tube in the other, and, with some asperity at being interrupted, asked the visitor what he wanted. This phase of his life was, however, of short duration, and was succeeded by another of perpetual professional and literary activity. It would be difficult to remember even a tithe of the work which he crowded into his days, or a tithe of the researches in which he was engaged. For some years he delivered lectures at his own house to medical men brought together by invitation, and these lectures were abundantly illustrated by experiments, often of a completely original character. So much were they appreciated by those who heard them, that they led to the presentation of a microscope and a purse of one thousand guineas from his professional brethren: a distinction probably unique in its kind, and one which Richardson would estimate at its true value.

Among his many pursuits, a favourite one was the study of anæsthetics, both local and general, and he introduced into practice the bichloride of methylene, which Sir Spencer Wells never deserted, and also the method of freezing the surface of the skin by ether spray, which soon passed out of favour, partly from the difficulty of making a clean incision in the frozen skin, partly from the ether having caught fire in more than one instance, so as to occasion consequences which were unpleasant, if not dangerous. The painless extinction of life occupied much of his attention, and stimulated him to many inventions, of which the "lethal chamber" at the Home for Lost Dogs is perhaps the only one which has survived him. He introduced into practice the very useful sodium ethylate, which has since found a place in the British Pharmacopœia, and did much to elucidate the action, both physiological and therapeutical, of the nitrite of amyl.

In 1875, at the Brighton meeting of the Social Science Congress, Richardson startled the public by his description of an imaginary "Hygeia," or City of Health, to be so constructed that disease should find no place within its boundaries, and that its inhabitants should die only from accident or from old age. He had then but lately joined the ranks of the total abstainers from alcohol; and the prohibition of the sale or consumption of this agent held a prominent place among the peculiarities by which the city

was to deserve its name. The scheme, it is hardly necessary to say, was in many of its features Utopian, and would have required a regeneration of the inhabitants, as well as the construction of special buildings fitted to contain them.

Prior to his sudden conversion to total abstinence, Richardson, although never intemperate, might have been described as a "jovial liver", and he was accustomed much to unbend at many social gatherings where wine and its kindred liquids were used without stint. He was a member of various clubs with droll names, mostly more or less Bohemian in their character, and was the life and soul of all of them, the frequent chairman or president of many. After his teetotalism, I saw less of him than before; but was forced to the conclusion that his change of habits had not been beneficial, nor do I feel able to acquit it of some share in bringing about a death which at least was premature. With his strong body and active brain, he might reasonably have been expected to attain his century.

Side by side with practice and with research, Richardson's literary activity was marvellous. Besides a romance called "The Son of a Star", and a vast number of contributions to medical and other journals and magazines, he wrote lives of Thomas Sopwith and of Sir Edwin Chadwick; originated and for a time edited the *Journal of Public Health* and the *Social Science Review;* and for twelve years before his death wrote the whole of the *Asclepiad,* a quarterly medical journal, of which nearly every number contained a biographical notice of some medical worthy of bygone times. He was also a frequent lecturer on social and scientific subjects, and a prodigious reader of general literature; loving especially, and often quoting from, the best writers of the Elizabethan and Stuart periods.

It must be apparent, even from this brief sketch, that Richardson's results in the way of permanent achievement were not commensurate with the energy which was expended on his work. A great deal of this work, indeed, as for example his popular lecturing, was in its very nature fleeting; his powers were spread over too wide a surface to produce the effects of more concentrated effort; and his intellect was not free from blemishes of a serious character. "The world little knows", says Faraday in a brilliant passage, "how many of the thoughts and theories which have passed through the mind of a scientific investigator have been crushed in silence and secrecy by his own severe

criticism and adverse examination; that in the most successful instances not a tenth of the suggestions, the hopes, the wishes, the preliminary conclusions, have been realised." This "crushing in silence and secrecy" was the very respect in which Richardson was deficient; and the deficiency led him to put forth views which did not bear the test of further investigation, and which only tended to bring his reputation for accuracy into discredit. He was evidently too prone to assume the truth of that which he wished to believe; and it is probable that his twenty years of campaign against alcohol failed to establish a single fact which was relevant to his views. Full of ingenuity, and of the "scientific uses of the imagination", he nevertheless was unable to subject this imagination to a curb of sufficient strength. He was prolific of hypotheses, but only a few of them have survived to become additions to the stock of recognised truth.

Whatever may have been the defects of Richardson's intellectual character, his personality was eminently interesting and loveable. *The Times* justly described him as "an admirable and original after-dinner speaker, a charming conversationalist and companion, and one of the most genial and amiable of men." From the same source I take the record that he received the honour of knighthood in 1893, and among other distinctions was a Fellow of the Royal Society, of the Society of Antiquaries, of the Royal College of Physicians of London, and of the Faculty of Physicians and Surgeons of Glasgow. He was M.A., M.D., and LL.D. of St. Andrews, Fothergillian gold medallist and past President of the Medical Society of London, President (for thirty-two consecutive years) of the St. Andrews Graduates Association, President of the Medical Temperance Association and of the Society of Sanitary Inspectors, and honorary physician to the London Temperance Hospital, the Royal Literary Fund, the Newspaper Press Fund, and the Society of Schoolmasters. "In no capacity", said *The Times*, "will his loss be more severely felt than as honorary physician and registrar of the Royal Literary Fund. Many men of letters owe much to his kindly intervention and advice."

On Wednesday, the 18th of November, 1896, an hour or two after the completion of his last book, Richardson was stricken down by apoplexy. He lingered until Saturday the 21st, and died without having recovered consciousness. He left a widow, two sons and a daughter.

The Ordinary Meetings of the Society are held at

11, CHANDOS STREET, CAVENDISH SQUARE,

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THE CHAIR IS TAKEN AT HALF-PAST EIGHT O'CLOCK P.M. PRECISELY.

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Ordinary Members are entitled to all privileges of membership, and to receive a copy of all the Society's publications issued during the year for which their subscriptions are paid. But Ordinary Members, resident beyond the limits of the Metropolitan Postal District, and having paid one or more annual subscriptions, may, on expressing a wish to that effect, be distinguished as Non-Resident Members.

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The Rules and Regulations of the Society, with an Exposition of its Objects, may be obtained from the Secretaries.

The Transactions for the Sessions 1859-60, 1866-67, *and* 1867-68 *are out of print. With these exceptions, those for the Sessions from* 1860-61 *to the last Session inclusive, may be obtained from the publishers,* Messrs. Shaw and Sons, Fetter Lane, E.C. *Prior to* 1859-60, *the Transactions were published in the* "Sanitary Review." *The issue of a New Series of the Transactions was begun in* 1882.

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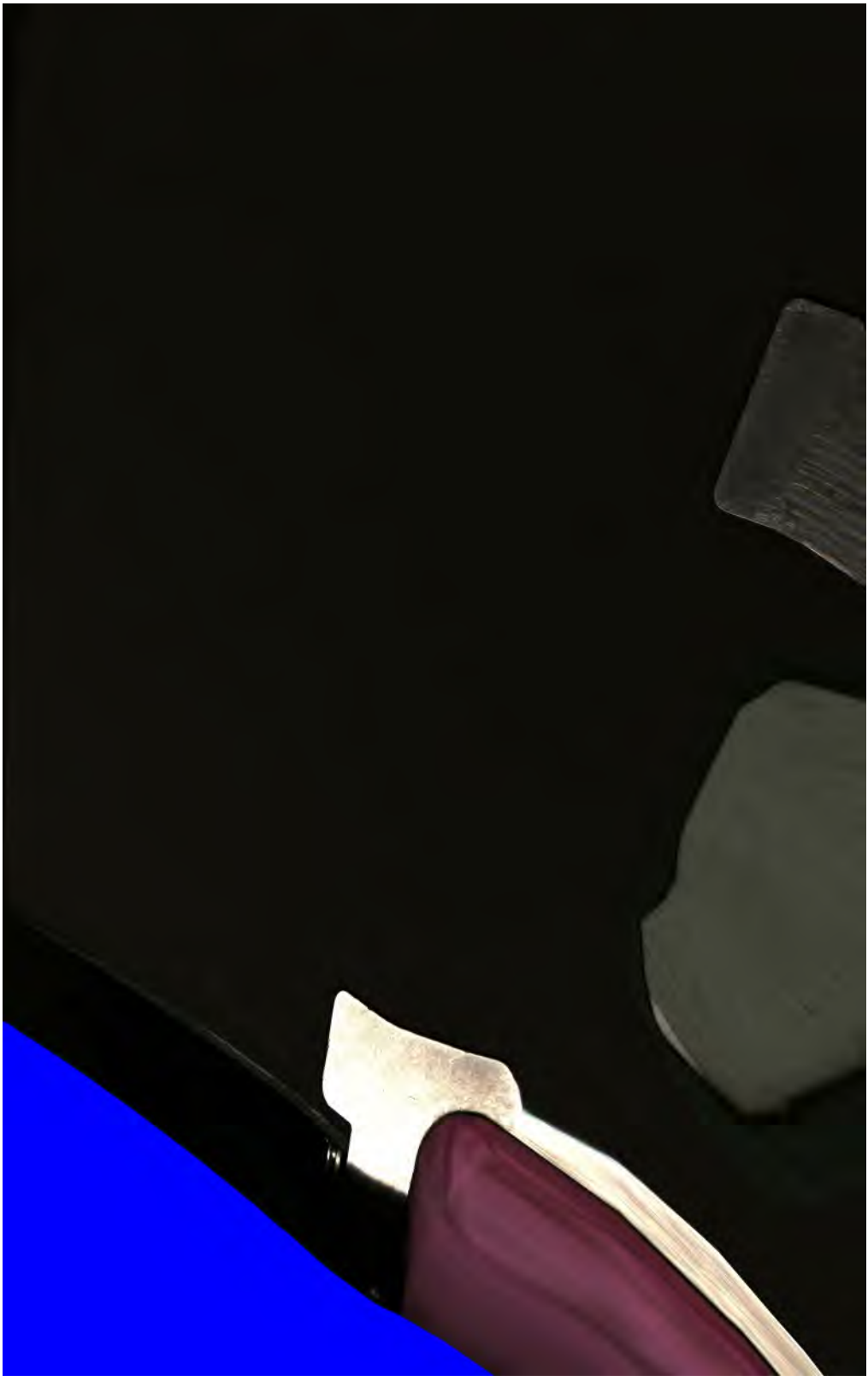
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